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A FIELD STUDY IN THE USE OF DIETARY AND URINARY VARIABLES  
IN DETERMINING OSTEOPOROSIS IN ELDERLY PEOPLE

by

Jane Steger Osborn

A thesis submitted in partial fulfillment  
of the requirements for the degree

in

MASTER OF SCIENCE

in

Nutrition and Food Sciences

UTAH STATE UNIVERSITY"  
Logan, Utah

1977

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Jane Steger Osborn

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## ABSTRACT

A Field Study in the Use of Dietary and Urinary Variables  
in Determining Osteoporosis in Elderly People

by

Jane Steger Osborn, Master of Science

Utah State University, 1977

Major Professor: Dr. Deloy G. Hendricks  
Department: Nutrition and Food Sciences

Three-day dietary records were analyzed for nutrient content and 24 hour urine samples were analyzed for calcium, phosphorous, total nitrogen, and free alpha-amino nitrogen for 210 elderly people. Dietaries and urine samples were collected twice, October and March at five month intervals, for each subject. Increases were found in both dietary intake and urinary components October to March. Based on a criteria of high dietary protein, low dietary calcium, high urinary nitrogen and low calcium, 23 subjects were selected as osteoporotic and 25 were selected as non-osteoporotic. This method of prediction was not supported by radiological evaluations.

Bone density and percent cortical area of the second metacarpal and the trabecular pattern of the femoral head were evaluated for each subject. A negative correlation of trabecular pattern with age indicated a general loss of bone with age.

Decreased percent cortical area was the most consistent bone phenomena associated with osteoporosis. No significant difference was found between sexes in any of the radiological analysis.

The osteoporotic condition is more closely associated with a loss of bone quantity than decreased bone quality. As yet, osteoporosis is not associated with specific nutrient(s) consumption or urinary excretion(s).

(91 pages)

## INTRODUCTION

As average life expectancy increases, new problems and unsolved mysteries of the aging process are revealed. Elderly people are products of and are affected by nutritional adequacy and its bearing on health and disease. As these people are more subject to disease their nutritional status and food habits become increasingly important to their longevity and enjoyment of life.

Osteoporosis is one disease more prevalent in elderly people than any other single age group (34). It is a metabolic disease in which normal osteoclastic activity exceeds osteoblastic activity and results in decreased bone strength and increased fragility. It is not a disease of acute onset but is progressive in its development. By the time it is radiologically detectable, up to 50 percent of the bone mass may have been lost (52). While nothing has been found to reverse the effects of bone resorption, dietary habits may influence its onset and severity (33).

Elderly people are frequently the victims of poor eating habits. For various reasons, psychological or physiological, milk, one of the main sources of dietary calcium, is often omitted from their diet. Other dietary sources of calcium are rarely substituted.

In studies involving calcium and protein, varying levels of dietary calcium could not stay the calcium-eliminating effects of high protein

intakes (31). The American diet, notoriously high in protein, may in this respect alone contribute to the development of osteoporosis.

Elderly human subjects in Southern Utah have been found to have high dietary protein intakes, while calcium has been low in many cases. These individuals may be considered a high-risk group for osteoporosis if their food habits have been prolonged over a lifetime.

The purpose of this field study was to identify osteoporotic individuals from this population through dietary and biochemical analyses and to obtain radiologic confirmation of the osteoporotic condition.



## LITERATURE REVIEW

### Bone Metabolism

Bone is a calcified collagenous matrix laid down by osteoblasts (12). Bone serves as the body's framework and the storage organ for minerals and buffers (7,15), with 99 percent of the body's calcium in the skeleton, approximately 17 g calcium per 100 g bone (38).

Once formed bone tissue is not static, but dynamic, continually undergoing apposition and resorption (53), or remodeling (12,14). Bone apposition continues throughout the lifetime at the subperiosteal bone surface (17,18,20) where bone is added longer at a greater rate in men than in women, resulting in larger bones (17). The endosteal surface undergoes loss-gain-loss throughout the years. For the first years of life the marrow cavity enlarges. Apposition begins during adolescence, continuing through the fourth decade in both sexes. After age 40, endosteal resorption begins and continues through the lifetime, with maximal losses occurring in the fifth and sixth decades (18,20).

It is the relative rates of gain at the subperiosteal surface and the proportional rates of loss-gain-loss at the endosteal surface that determine the 1) gross size of the bone, 2) amount of compact bone, and 3) proportion of compact bone, which in turn determine the mechanical properties of resistance to compression, bending, breaking, and radiological appearance of bone (17).

The major bone dimensions are 1) total subperiosteal width, 2) medullary cavity width, and 3) cortical thickness. From these, defects in total bone formation (decreased subperiosteal width), or excess endosteal surface resorption (increased medullary width) can be distinguished (19).

There are two types of bone, compact or cortical, and cancellous or trabecular. Trabecular bone comprises one fifth of the total bone mass but is narrowly distributed; one half in the femur, tibia, and humerus; one third in the vertebral column; and one sixth throughout the rest of the skeleton. Trabecular bone has a larger surface area: volume ratio than compact bone and is more exposed to sudden metabolic demand (15). In bone atrophy, then, the removal of a single "layer" from the compact bone results in a smaller percentage thinning than the removal of a trabecular layer (8).

### Osteoporosis

Osteoporosis is probably the most common metabolic disease of the skeleton in man. Long-standing controversies exist concerning its cause and course of development, as to whether diminished bone is due to insufficient matrix, osteoblastic activity, deficient bone formation, or excess resorption (23). The bone loss of osteoporosis develops over a long period of time (25) and may be the end result of many processes, not a singular disease (15).

### Definition

Literally "porous bone" (19), osteoporosis is considered by some to be an irreversible disease (24) of bone tissue quantity and not

quality (52), a reduction in the amount of bone without a change in its chemical composition (40). Yet others do not consider it a disease but a common metabolic disorder (12).

Osteoporosis may be recognized absolutely as the decrease in bone mass, which is radiologically recognizable by a decreased radiodensity (5) or relatively, as a diminution of bone mass per unit volume as compared to normal (13,45). Functionally, osteoporosis is a decrease in the total bone mass below the level required for mechanical support (24) or maintenance of the structural function of the bone as a framework for the body (15).

#### Characteristics

Aging of the bone involves quantitative and qualitative changes (8) that may be localized or generalized throughout the entire skeleton (52). Because osteoporosis does not affect all parts of the skeleton at equal rates (25), the diminution of bone density may be more apparent in X-rays of the axial skeleton in the biconcave vertebra compression -because the percentage of trabecular bone is greater than in the long bones (2,7,52).

There are no distinct biochemical features (29) or specific clinical laboratory findings (51) relating to osteoporosis, making the identification bone-dependent. Involving both trabecular and compact bone (42), osteoporosis affects the strength of cancellous bone more than that of compact bone (8). Progressive cortical thinning (12,52) results in a nearly hollow shell (18), decreased volume and increased porosity (8). Morphologically, the trabeculae are thinner, fewer, and larger than normal (12,14,15).

Because both matrix and mineral are lost (18) osteoporosis is characterized by diminished bone mass without a change in chemical composition (11), skeletal atrophy, or thin bones, but an otherwise structurally normal skeleton (23).

As bone mass diminishes relative to bone volume, mechanical strength decreases, vertebrae are less resistant to compression, and tubular bone and the femoral neck are less resistant to bending stress (18) resulting in femoral neck fractures, fractures of the distal radius and ulna, and spontaneous vertebral collapse (16).

### Fractures

The consequence of less-than-normal bone mass is an increased incidence of fracture (35). With the diminished mass of cortical bone and supportive cancellous structures, the femoral neck is especially vulnerable to fracture and may be ranked as an important indirect cause of death (8,18). The incidence of vertebral fracture has been found to be proportional to bone mineral content (48) which, along with femoral neck fractures, may suggest skeletal wasting and bone atrophy, rather than severe trauma (8). Characteristic fractures are generally of a crushing and deforming nature, making reconstruction of the original structure difficult or impossible (52).

Fracture patterns were found to be age-specific (8) and to exhibit characteristic curves when plotted age versus incidence. The pre-wage-earning pattern showed a large incidence in both sexes in youth, with a rapid fall to smaller numbers in middle and adult life, exhibiting an "L" curve. The male:female ratio was 2:1, and the bones most frequently affected were the tibia shaft and clavicle.

The wage-earning pattern (8) showed a high incidence in men during working ages with a low incidence in youth, old age, and women. The characteristic curve resembled an "A." The male:female ratio was 3.3:1, and the most common fractures occurred in the phalanges and metacarpals.

The post-wage-earning pattern (8) showed a slight bias toward young men, and a rise in both sexes in old age, especially in women. Characteristically a "J" curve, the male:female ratio was 1:1.2, and the bones most frequently involved were the femur, pelvis, and upper humerus.

Also found was a sharp increase in fractures in women, which by the ages of 70-79 was four times greater than in men. Under the age of 50 fractures in men were three times greater than in women. After age 50 the rate was reversed. Men of age 80 had a liability of fracture of the femoral neck 30 times as great as men under 40, while women of 80 had a liability of femoral neck fracture 300 times as great as women under 40 (8).

#### Determination

The early diagnosis of osteoporosis is radiologically dependent (35). Visual assessment of skeletal mineralization from x-rays is not a sensitive indicator of demineralization (10,13) and the evaluation of a single exposure is considered insufficient unless considerable bone has been lost (1,19,17,35). However, it is diagnostically and functionally more important to consider the absolute amount of bone still present than the fraction of bone lost (35). Estimates as to the amount of bone lost before detection vary from 15 to 50 percent (11,12,34,52).

For the initial diagnosis the loss of fine trabeculae (53), thinning cortices (12), and increased medullary cavities (18) all give the appearance of decreased mineralization. Quantitation of bone density is best determined by caliper measurement and densitometry (12,26,35).

In measuring the calcium content of bone from x-rays, there is a problem in determining the contribution of the soft tissue to total density (15,38). Two techniques,  $^{125}\text{I}$  radiation densitometry and hand immersion in water during x-ray exposure (38) have attempted to minimize this effect.

### Epidemiology

#### Person characteristics

Age. Universally, skeletal weights decrease with age and most, if not all, bone loses mass with age (18). Bone loss is a normal aging phenomenon (1,26,35), beginning about the fifth decade in both men and women (5,8,21). As life expectancy increases, involutional changes occur, one of which is bone atrophy, resulting in fragile, brittle bones in the aged (8). Osteoporosis may be rare in some countries due to the low age of life expectancy (42).

Whether bone measurements are expressed as cortical thickness, cortical thickness:bone width ratio, or as percent cortical area, women and men lose bone at a faster rate than it is deposited after age 40 (21). According to Garn, et al. (21) the bone loss after age 50 approximates 15 percent in men (3 percent per decade) and 30 percent (8 percent per decade) in women. The magnitude of the loss is such that by age 80

the total bone mass in women is significantly less than that of normal women before age 45, resulting in two distinct populations (35).

If osteoporosis is at all attributable to prolonged negative calcium balance age may also affect osteoporosis as it affects the duration of a negative calcium balance (11).

Generally considered a condition of old age, osteoporosis has been reported in juveniles, characterized by a relatively rapid onset of bone loss about the ages of 8 to 15, continuing for one to four years, but followed by a large degree of skeletal restoration (29).

Sex. Osteoporosis has been long-considered a phenomenon of older women, hence the term "post-menopausal osteoporosis." It was considered to be restricted to a relatively few, inactive, post-menopausal, white women with low milk consumption (21). Bone loss has now been described in both sexes in all populations studied (18).

Osteoporosis is more common in women than men, occurring earlier, with greater frequency, and to a greater extent (5,7,15,21,37). The magnitude of bone loss from age 30 to 80 is absolutely and relatively larger in women than in men. Reportedly the loss approximates 750 g from a 3 kg body skeletal mass in women and 450 g from a 4 kg skeletal mass in men (17,18).

Ovarian function may affect skeletal mass. Ovariectomized women have shown early signs of bone loss, losing the same amount of bone as normal menopausal women, proportionately earlier (18). Repeated pregnancies may drain body calcium due to fetal development and lactation, but Albright (2) did not consider this an important factor in the onset of osteoporosis.

Race. Smith et al. (50) found an age-associated difference between two racial groups in cortical thickness and external diameter. Comparing blacks and Caucasians, external diameters increased with age in blacks and whites while cortical thickness diminished 32 percent in whites and 26 percent in blacks, from the youngest to the oldest.

The percent of white women subjects in the lowest two of four grades of relative vertebral density increased from 39 percent in the 45 to 54 age group, to 77 percent in the 65 to 74 age group, to 90 percent in the 75 year-and-older age group. No comparable figures were reported for blacks in the study (50).

It appears that some races are protected against bone loss, relatively (21), but skeletal weight has been found to decrease in both blacks and whites, with blacks having a larger bone mass at all ages (17). In a U.S. population, black women were found to have a lesser rate of bone loss than their white counterparts, while black men lose bone similarly to white men (18). Those of African heritage have higher metacarpal cortical thicknesses, fewer hip fractures, and a lower incidence of spinal osteoporosis than whites (17,42,50).

Associations have been suggested between lactase deficiency and osteoporosis. However, in blacks they are inversely related, the incidence of lastase deficiency is high, and the incidence of osteoporosis is low (4,6).

Socio-economic status. To a degree calcium consumption increases with socio-economic levels and may influence a greater incidence of osteoporosis in low socio-economic areas (55) although threr have been no reports to bear this out. As economic status affects bone rates,



relative remodeling rates and skeletal size are a reflection of availability of calories and nutrient metabolism (17).

Initial amount of bone. An effect of the amount of bone on the rate of development of osteoporosis has been seen. For example, taller individuals lose less bone with age than do shorter individuals (21), women lose a greater percentage of their bone mineral content than men, due to a smaller skeleton, and blacks, with larger bone masses at all ages (11), lose bone to a lesser degree than whites (20). With an initial large bone mass, more bone can be lost before appearing abnormally weak (35).

Smith et al. (48) estimated that subjects with vertebral fracture lose bone at the same rate as controls, giving no support to the theory that osteoporotics constituted a sub-population. The bone mineral content for all ages was normally distributed, and it was concluded that the bone mineral value for osteoporotics was at the lower end of the distribution.

#### Incidence and prevalence

Little information is available statistically on the incidence and prevalence of osteoporosis. Not a communicable disease, nor easily diagnosed, statistics are rarely calculated or required to be kept by health departments and/or hospitals. Much of this information is available only by extrapolation and estimation.

Radiologically evaluated, vertebral atrophy of a substantial degree occurs in more than 50 percent of women over 45 in the U. S. Using this estimate, it was estimated from the 1963 census for people over 45 that 14 million women had a significant amount of vertebral wasting (50).

For people over 50, the estimated morbidity of osteoporosis was greater than 20 percent in 1967 (6). There is also a loss of tooth-bearing bone with age, which may be considered a manifestation of bone loss. Israel (28) estimated that more than 50 percent of the U. S. population is edentulous by the age of 60.

The universality of bone loss was demonstrated by Garn et al. (21) in a comparison of three countries: the U. S., Guatemala, and El Salvador. Both sexes in these countries had age-associated cortical bone loss, beginning about the fifth decade. Women lost more bone than men, and men and women both lost bone after age 40.

The prevalence of diminished bone density is reportedly increased in low fluoride areas in men and women. Fluoride consumption apparently decreases this prevalence in women significantly, with the same trend being seen in men, but insignificantly (5). Osteoporosis has been reported twice as frequent in women in low fluoride areas as in women from high fluoride areas (25).

### Etiology

There are many etiological theories proposed for osteoporosis. The only agreeable point is that its etiology is multifactoral (5,24). Current theories include aging (8), hormonal changes and balance (2,55), immobilization (6,24,25,51,52), and nutrition (40,41,42,50,55).

Senescence. As other manifestations of aging are not preventable (18), all people lose bone with age (39), and bone loss is considered a physiological change (12), there appears to be little justification

for distinguishing post-menopausal and senile osteoporosis from bone loss with age (21). Hormonally, women are affected earlier than men, but as the hormonal levels are reduced, men also are affected by osteoporosis (12).

Hormonal. The post-menopausal state has long been considered the most common etiological factor in the development of osteoporosis (2). It is difficult to deny the overwhelming evidence associated with bone loss at the onset of menopause. This association, however, does not constitute a cause, although many insist that osteoporosis is the inevitable consequence of gonadal failure and decreased estrogen production (12,20). This does not explain the slower bone loss seen in men. Men are then spared due to continuing androgen production (12), but eventually the androgen levels decrease and they, too, are affected by osteoporosis.

Garn et al. (21) deny that bone loss is due to menopausal onset, reasoning the bone loss occurs at similar ages in women with delayed onset of menopause. Additionally, more bone is claimed to be lost than is accountable for by simple estrogen withdrawal (18). There is no direct evidence that a lack of estrogen causes osteoporosis.

Immobilization. Notable bone loss is seen in severe immobilization. Some immobilization effects may be overcome by weight-bearing stimulus and activity. Therapy for bone atrophy thus caused is an increase in energy expenditure, but efficacy in cases of osteoporosis is doubtful (18). It is doubtful that the degree of inactivity associated with aging is singularly responsible for bone loss.

Nutritional. Nutritional factors have also been implicated in the development of osteoporosis. Diet is not considered the predisposing factor (18). A low calcium intake is to be expected in association with a decreased intake of all nutrients and should not be considered a unique nutrient lack. Osteoporosis has been reported to occur concomitantly with protein-calorie malnutrition (21). Vinther-Paulsen (54) reported that the incidence of osteoporosis among 33 subjects with less than 500 mg calcium daily was 75 percent, compared to a 15 percent incidence among those with calcium intakes greater than 500 mg daily.

As ingested fluoride concentrates in the hard tissue, increasing linearly with increased fluoride in drinking water up to 4 ppm (46), oral fluoride treatment may inhibit osteoporotic bone loss (16) by increasing the stability of the bone mineral against resorption (24). In a six month balance study of fluoride's effect on calcium, urine calcium was reportedly decreased initially, but after six months urinary calcium returned to normal. Rich et al. (47) concluded that calcium balance may be altered on an individual basis as may retention, and that 1 mg fluoride daily would increase calcium retention but that there is as yet no information as to a minimum dose.

Bernstein et al. (5) reported the prevalence of diminished bone density was increased in low fluoride areas in both sexes. Determining whether prolonged ingestion of small amounts of fluoride has an effect on bone density and the rate of the progression of osteoporosis, their results indicated that fluoride at 4.0-5.8 ppm levels in water significantly reduced the prevalence of osteoporosis. Fluoride consumption apparently decreased the prevalence of reduced bone density in men and women.

Western regional studies found no difference in bone density between fluoride and non-fluoride areas, but a significant difference was seen in the reduction of dental caries (44).

One possible cause of bone loss is resorption due to a negative calcium balance. Associated with this, people may be unable to adapt their calcium requirement to their intake, resulting in bone loss. After 10 days on low calcium diets, normal subjects' urinary calcium decreased by approximately 30 percent, while those with osteoporosis showed little or no decrease or adaptation to stress conditions. Nordin (40) considered the difference in calcium intake between osteoporotic subjects and controls significant if not associated with a corresponding change in urinary calcium excretion. He contends that a continued negative calcium balance results in mineral resorption and rapid removal of matrix, and that the relative hypercalciuria described is more likely the cause of osteoporosis than the result.

Two hypotheses concerning the development of osteoporosis by Nordin (42) propose that osteoporosis is 1) a primary change in bone matrix leading to inevitable bone breakdown, or 2) that there are nutritional implications of calcium and protein, especially a negative balance. In many parts of the world diets are very low in calcium without any apparent ill-effects.

In one study (9) six males were subject to varying levels of protein on a purified protein diet of 0.9, 12.0, and 24.0 g nitrogen daily, with 100 mg calcium. After 60 days, a direct relationship was found between urine calcium and protein intake. Urinary calcium

increased with increased protein level, as did calcium absorption. The net result was a decrease in calcium retention.

Linkswiler et al. (31) also tested the effect of dietary protein on calcium retention and absorption in 33 young adult males at varying levels of protein, 47, 95, and 142 g daily, and varying levels of calcium at 500, 800, and 1400 mg daily. On the low protein diets, positive calcium balance was unaffected by the level of calcium in the diet, while at the 95 g protein level, calcium balance was first maintained at the 800 mg calcium level. At the 142 g protein level, positive calcium balance could not be maintained at either the 500 or 800 mg calcium levels, and only three subjects had positive calcium balance at the 1400 mg calcium level. Calcium absorption was unaffected by protein intake at 500 mg calcium, but increased at 95 and 142 g protein and 800 and 1400 mg calcium. The absorption effect of protein on calcium was calcium-dependent.

Urinary calcium was significantly affected by the level of protein in the diet. Regardless of the level of calcium, less calcium was excreted on low protein diets. Calcium retention was significantly affected by protein at all calcium levels, being greater at low protein levels.

Brink (7) suggests that the lesser ability of the skeleton to retain calcium may be the result of the dissolution of bone salts for buffering acid loads in the body. High protein intakes increase the acid load of the body and therefore the amount of phosphorus in bone salts needed for neutralization is increased.

In five female and twelve male subjects calcium retention increased positively as calcium intake increased. Finding no correlation between dietary and urinary calcium, Nordin (41) concluded that the stability of urinary calcium may accentuate a negative calcium balance at low intakes, and may contribute to the development of osteoporosis by failure to adapt urinary calcium.

Osteoporosis has been associated with low dietary calcium in humans. If this association holds, the effects of a high-calcium intake would be to inhibit the primary symptoms of calcium depletion in the skeleton.

Harrison et al. (23) assumed that if osteoporosis was due to a calcium deficiency, there would be an increase in the body's avidity for calcium, whereas if it was due to defective osteoblastic activity the body would have little affinity for the increased calcium. To test this, 16 subjects with osteoporosis, 60 years of age and older, were supplemented with calcium in their diets. Osteoporotics consuming the high-calcium diet went into strongly positive calcium balance, suggesting to the authors an increase in absorption and deposition, and decreased resorption, presumably refuting the defective osteoblastic theory. Abnormal calcium metabolism that was seen in 50 percent of those with osteoporosis, was explained as due to a different stage of the condition or a different form. This possibility has not been supported or recorded elsewhere.



## Therapy

Because once structural failure occurs it is impossible to increase the amount of bone (35,39), it is important to recognize and evaluate the preventative and therapeutic value of any agent that affects the progress of osteoporosis (5,25).

With no known cure or prevention, symptoms of osteoporosis may be relieved but not reversed (23).

Hormones. Since Albright (2), hormones have been the central and current attempt in prevention and therapy for osteoporosis (12,35,49). Claims for the efficacy of hormonal treatment vary from supplementation that may delay the onset of osteoporosis up to 10 years (12) to the failure of prolonged treatments to heal or alleviate the condition (30).

Fluoride. Optimal levels of fluoride for the prevention of osteoporosis are unknown (25). It appears reasonable to expect that fluoride could have similar effects on bone as it has on teeth (16,25) but the value of this preventative therapy must be considered in light of the length of time continued and the stage of development that the fluoride ingestion was begun (5).

Calcium. The thrust of all osteoporotic therapy is to decrease the loss of calcium from the body. Some therapies may be circumspect in their approach such as fluoride and hormones. Direct calcium therapy to increase absorption, retention, and deposition have also been employed. The most that can be said in favor of this use is that insofar as it may inhibit resorption it is considered beneficial (10,25). There is no convincing radiological evidence of improvement (31), but



it must be remembered that a condition that has developed progressively over many years cannot be alleviated or cured in a few months.

## METHODS AND PROCEDURES

### Experimental Design

Two hundred ten ambulatory southern Utah elderly people kept dietary records for three days, and collected urine samples for 24 hours in October 1974 and March 1975. Based on urinary excretions of total nitrogen and calcium, and dietary protein and calcium, 23 people considered most likely osteoporotic (high urinary total nitrogen and calcium, high dietary protein and low dietary calcium) and 25 people considered least likely osteoporotic (low urinary total nitrogen and calcium, low protein and high dietary calcium) were selected for x-ray exposures.

### Data Collection

#### Dietary

In October and March elderly human subjects were asked to keep complete three-day dietary records. On day four, subjects submitted their records and were interviewed to clarify food descriptions and quantities. In an effort to improve the effectiveness of these interviews, food samples were used as references in the March sampling.

#### Urine

Twenty-four hour urine samples were collected during one of the days dietaries were kept. Total volume and specific gravity, determined

by a refractometer, were recorded for each 24-hour sample. One hundred fifteen ml aliquots were acidified and refrigerated in polyethylene bottles for later analysis.

### X-ray

Men and women were selected for x-rays on the basis of estimated dietary protein and calcium consumption and urinary calcium and total nitrogen excretion.

Two exposures of each subject were used. An aluminum stepwedge was included in each exposure of the metacarpals for radiodensity standardization. The stepwedge ranged from 1 mm to 21 mm aluminum in 0.5 mm increments. Right and left hands were exposed together, palms down, fingers extended and separated. The stepwedge was located generally parallel to the fingers, between the hands.

Femoral neck exposures were frontally viewed.

### Analysis

#### Dietary

Dietary records were computer analyzed for estimated nutrient content according to U.S.D.A. Handbook 8 (56), and printed as a three-day average.

#### Urine

Urine was analyzed for total nitrogen, free alpha-amino nitrogen, calcium, and inorganic phosphorus.

Total nitrogen. Total urinary nitrogen was determined by a modified micro-Kjeldahl method of the Association of Official Analytical Chemists (3). One ml samples were digested with sulfuric acid using Helgar granules as boiling chips and catalysts, and sodium sulfate. Samples were distilled into a boric acid solution with a bromcresol green indicator, and titrated to the endpoint with hydrochloric acid of known acidity.

Free alpha-amino nitrogen. Free alpha-amino nitrogen was determined colorimetrically by the method of Lorentz and Flatter (32) as a possible indication of protein metabolism. Fifty  $\mu$ l urine were mixed with 1.0 ml phosphate buffer (0.5 M in dimethylsulfoxide). After 30 minutes excess p-benzoquinone was extracted with one 5 ml portion of diethyl ether. After aspirating the ether layer, the color-complex solution was diluted with 2 ml distilled water to a workable volume. Colorimetric values were determined within 60 minutes of dilution at 492 m $\mu$  on a Bausch and Lomb Spectronic 20.

Inorganic phosphorus. Urinary inorganic phosphorus was determined colorimetrically by a modified method of Gomori (21). Urine samples were filtered to minimize sediment interference. Fifty  $\mu$ l filtered samples were combined with 5 ml "MS" aqueous solution (1 percent magnesium chloride, 1 percent ammonium molybdate, 2.8 percent sulfuric acid) and 0.25 ml "elon" (1 g p-methylaminophenol sulfate in 100 ml 3 percent sodium bisulfite). Colorimetric determinations were made after 45 minutes at 700 m $\mu$  on a Bausch and Lomb Spectronic 20.

Radiological. Cortical radiodensity was determined densitometrically. The radiodensity of stepwedges were determined first, providing

standards for each metacarpal exposure. Right and left hand cortical radiodensities were determined at midshaft on the second metacarpal. Bone radiodensity was determined by subtracting the baseline radiodensity measurement of the soft tissue from the maximum radiodensity of the finger, including soft tissue and bone.

Total diameter, medullary width, and combined cortical thickness expressed as percent cortical area were measured at the midpoint of the second metacarpal with calipers to the nearest whole millimeter using the method of Meema (34).

The osteoporotic condition was subjectively determined in the metacarpal visually by having the x-rays evaluated independently by two radiologists.

The trabecular patterns in the femoral neck were evaluated by the radiologists, and given a relative grading from one to six. A grade of one indicated the least amount of trabecular pattern, while a grade of six indicated a complete pattern. Standards for grading are in Appendix D.

Statistical. The data obtained in this study were analyzed statistically by correlation, analysis of variance, discriminant function analysis, and t-test.

Dietary and urinary data were correlated with themselves and each other to determine significant relationships.

Discriminant function analysis was used to predict groups association after the presence or absence of the osteoporotic condition was determined.

The t-test was employed to determine significant differences between dietary and urinary seasonal variations.

Statistical analysis of variance was used to determine significant radiological differences between osteoporotic and non-osteoporotic people and differences between sexes.

## RESULTS AND DISCUSSION

### Dietary

The dietary components of major interest were calcium, phosphorus, and protein.

### Calcium

Estimated mean calcium daily intake from the October sampling was  $633 \pm 289$  mg. In March the estimated mean calcium daily intake increased significantly ( $P < .05$ ) to  $726 \pm 324$  mg. The wide variation as indicated by the standard deviations is characteristic of free-living populations and individual variation.

The men and women involved in this study were part of a Senior Nutrition Aid Program. The purpose of this program was to evaluate the nutritional status of those involved and to help them improve their health through improved nutrition. In this general respect the participants were encouraged to change their dietary habits, but no specific recommendations were made in an effort to alter the specific nutrients herein considered.

Frequency distribution by sex showed that women consumed less calcium in October and March than men; the mode for October for men and women was the same; the mode interval for men increased from October to March while for women over the same period the mode interval decreased.

As diets were not analyzed by food groups, the observed changes in calcium intake cannot be explained by a change in food habits or preference. The increase of all dietary components from October to March may reflect a simple increase in caloric consumption.

### Phosphorus

Estimated mean phosphorus daily consumption was  $985 \pm 312$  mg in October. Reflecting an increase in food consumption, estimated mean phosphorus daily intake increased to  $1042 \pm 332$  mg in March. Frequency distribution by sex showed that more women cumulatively consumed less phosphorus than men at mode intervals; the mode intervals for men and women remained constant for both sampling periods. However, the mode interval for men was always the greater of the two sexes. Phosphorus consumption for men was redistributed toward more men consuming more phosphorus in March, with an increase of 18 percent at the highest mode interval. Phosphorus consumption of the women was redistributed more uniformly about intermediate intervals with a 5 percent decrease at the mode interval and a 5 percent increase at the highest interval. In March the range of phosphorus consumption increased for women. The overall increase in the population's estimated mean phosphorus daily intake was more dependent on the increase and frequency of the increase in men than in women. Differences in distribution characteristics between men and women may indicate a difference in food patterns between the sexes.



### Protein

Consistent with other dietary nutrients, estimated mean protein daily consumption increased significantly ( $P < .05$ ) October to March, from  $58.7 \pm 17.6$  g to  $63.9 \pm 16.0$  g. Frequency distribution by sex showed that protein consumption of men was greater than that of women in October, and increased to a greater extent in March. Mode intervals for women remained the same both seasons, and decreased slightly in frequency from October to March. The mode interval for men increased from October to March as did the frequency at the interval. While the range of protein consumption remained constant for women October and March, the range of protein consumption for men increased in March. Both sampling periods the mode interval for men was greater than the population mean. In October, the mean and mode interval was the same for men and women, but in March the mode interval of women was less than that of the mean. Thus the protein consumption of the men accounted for the greater part of the increased estimated mean protein daily consumption October to March.

The similarity between the phosphorus and protein distributions suggests a relationship may exist in the consumption of these nutrients. One explanation is that foods high in protein are generally also high in phosphorus. As the protein consumption was high both in October and March, this association would partially account for the increased phosphorus intake.

### Calcium:phosphorus ratio

The relationship of dietary calcium and phosphorus was determined by a ratio of the two. The ratio is commonly expressed with calcium

at unity. In October the mean ratio was 1:1.58, estimated mean phosphorus daily intake being 58 percent greater than that of calcium. In March the mean ratio was 1:1.47. The change indicated a relative decrease in estimated mean phosphorus daily consumption with respect to calcium. Table 1 summarizes the dietary changes observed.

Table 1. Total population variation of selected mean daily dietary nutrient intakes, October to March

Nutrient	Mean $\pm$ S.D.		Percent increase
	October	March	
calcium, mg*	633 $\pm$ 289	726 $\pm$ 324	10
phosphorus, mg	985 $\pm$ 312	1042 $\pm$ 332	6
protein, g*	58.7 $\pm$ 17.6	63.9 $\pm$ 16.0	9
calcium:phosphorus	1:1.58 $\pm$ 5.88	1:1.47 $\pm$ 4.90	7

\* Significant at 5 percent level

### Correlations

Table 2 summarizes the correlation coefficients generated in correlation matrices for dietary nutrients only. Consistent with the estimated mean daily dietary intake increases October to March was the tendency for the correlation coefficients to increase seasonally. Significant relationships were found between phosphorus and calcium, protein and calcium, and phosphorus and protein. The latter supports the protein-phosphorus association in high-protein foods.

Table 2. Summary of correlation matrices of selected dietary nutrients for total population

Nutrients	Correlation coefficients		
	October	March	Combined
phosphorus, calcium	.78	.90	.84
protein, calcium	.56	.66	.62
protein, phosphorus	.81	.87	.84

### Urinary

So much greater was the increase of excretion of urinary components October to March as to be unexplained as simple reflections of the dietary changes.

### Calcium

The very significant ( $P < .01$ ) increase in calcium excretion October to March was  $54.8 \pm 79.1$  mg to  $99.4 \pm 117$  mg. Frequency distribution by sex showed that mode intervals were the same for men and women in October; increased for both sexes in March, more so for men than for women. The range of calcium excretion increased by two intervals for men in March. This maximum distribution range was exhibited both seasons in the women. The frequency of the mode interval fell considerably for both sexes from October to March. The high frequency of lower excretion values in both sexes does not support the association of osteoporosis and excess calcium excretion or loss.

### Phosphorus

Mean phosphorus excretion increased very significantly ( $P < .01$ ) from  $485 \pm 297$  mg in October to  $1080 \pm 755$  mg in March. Frequency distribution by sex showed that the mean and mode intervals were the same for men and women in October. In March the mode intervals increased for men, to exceed the mean interval value, while for women the mode interval also increased, but not to equal the mean. In March, 51 percent of the women were redistributed to create a new high interval of phosphorus excretion. The lowest interval of the October sampling for women was not seen in March. The distribution range of men showed no such change.

### Total nitrogen

Mean total nitrogen excretion increased very significantly ( $P < .01$ ) from October to March,  $5.1 \pm 3.0$  g to  $8.6 \pm 5.8$  g. Frequency distribution by sex showed that the mode and mean interval for men remained the same both samplings, while the mode interval for women increased to the mean interval in March. Distribution ranges were the same for both sexes both seasons; however, in March none of the population fell into the lowest interval. Three percent of the men created a new high interval in March, while 5 percent of the women created two consecutively higher intervals in March. The range of total nitrogen excreted was then greatest for the women of March's sampling. The mean and mode interval for mean did not change seasonally, but the frequency diminished slightly in March. The mode interval for women increased to the mean interval in March while also increasing in frequency.

### Free alpha-amino nitrogen

The greatest seasonal change occurred in free alpha-amino nitrogen excretion. Mean excretion increased very significantly ( $P < .01$ ) October to March, from  $94.8 \pm 63.2$  mg to  $214 \pm 122$  mg. This increase is consistent with the increase of total nitrogen excretion. The October mean free alpha-amino nitrogen accounted for 2 percent of the mean total nitrogen excreted. In March, this increased to 2.3 percent indicating a relative and absolute increase of free alpha-amino nitrogen. Frequency distribution by sex showed that the mode intervals for men and women increased in March. The October mode intervals for men and women were less than the mean interval, while for males only, the March mode interval was greater than the mean interval. The range of free alpha-amino nitrogen excretion increased in March for the women, but remained the same for men both seasons.

Table 3 summarizes the changes in urinary excretion seasonally.

Table 3. Total population variation of selected urinary excretions, October to March

Urinary excretion	Mean $\pm$ S.D.		Percent increase
	October	March	
Calcium, mg**	$54.8 \pm 79.1$	$99.4 \pm 117$	81
phosphorus, mg**	$485 \pm 297$	$1080 \pm 755$	123
total nitrogen, g**	$5.1 \pm 3.0$	$8.6 \pm 5.8$	69
free alpha-amino nitrogen, mg**	$94.8 \pm 63.2$	$214.2 \pm 122.9$	127

\*\* Significant at 1 percent level

### Correlations

In Table 4 the correlation coefficients from the correlation matrices for urinary components are summarized. The most significant relationships were found between phosphorus and total nitrogen, phosphorus and free alpha-amino nitrogen, and total and free alpha-amino nitrogen. Nitrogen cannot be stored in the body, and body phosphorus is controlled by urinary excretion rather than absorption. In this light, a diet adequate or high in protein would be expected to show as high urinary nitrogen, and the excess phosphorus associated with that protein would also appear in the urine. The lesser relationship between calcium and phosphorus, and calcium and nitrogen may be attributed to the different mechanisms of control. Body calcium is controlled by absorption, which is adversely affected by the presence of phosphorus as phytate and other chelating agents. Unabsorbed calcium excreted in the feces would be greater than urinary calcium excretion on a high protein-high phosphorus diet due to the inhibiting effect of phosphorus.

Table 4. Summary of correlation matrices of selected urinary excretions for total population

Urinary excretions	Correlation coefficients		
	October	March	Combined
phosphorus, total nitrogen	.66	.79	.80
phosphorus, calcium	.46	.39	.44
phosphorus, free alpha-amino nitrogen	.67	.87	.87
total nitrogen, free alpha-amino nitrogen	.64	.77	.78
calcium, free alpha-amino nitrogen	.38	.36	.41
calcium total nitrogen	.36	.34	.38

Table 5 summarizes the correlations of dietary and urinary components. The insignificance of these correlations may be due to the urine collection timing relative to the dietary recording. The dietary values are the averages of three days' intake, whereas the urine samples are a single 24-hour collection. Some urine samples may not have been for the full 24 hours, and their collection was not the same with respect to dietaries for all individuals, varying from concurrent to 24-48 hours previous to the last day of the dietary records.

Table 5. Summary of correlation matrices of selected dietary nutrients and urinary excretions for total population

Variables		Correlation coefficients		
Dietary	Urinary	October	March	Combined
calcium	total nitrogen	.01	.19	.17
calcium	calcium	-.02	.13	.11
calcium	phosphorus	.01	.19	.19
calcium	free alpha-amino nitrogen	-.06	.17	.16
phosphorus	total nitrogen	.07	.16	.15
phosphorus	calcium	-.04	.10	.06
phosphorus	phosphorus	.07	.19	.17
phosphorus	free alpha-amino nitrogen	-.02	.17	.13
protein	total nitrogen	.13	.16	.19
protein	calcium	-.02	.05	.05
protein	phosphorus	.04	.16	.17
protein	free alpha-amino nitrogen	.01	.16	.16

### Radiological

The selection of the subpopulation for radiological evaluation was based on dietary nutrient and urinary excretion spectrum extremes, anticipating that diets high in protein and low in calcium would effect a net negative calcium balance in the body, thus contributing to a possible osteoporotic condition. Osteoporosis was determined to exist in those individuals who both radiologists evaluated to have lost significant metacarpal cortex. Travecular pattern indices were evaluated but were not significantly associated with metacarpal loss. From the rate of success of this criteria, protein, calcium, and nitrogen excretion are not sufficient for diagnosing osteoporosis. Only five of the subjects selected as most-likely osteoporotic were radiologically confirmed. Fourteen selected as least-likely osteoporotic were radiologically determined to be osteoporotic.

Of the men and women selected, radiological evaluations did not reveal any significant differences in trabecular pattern index (TPI), metacarpal radiodensity, or percent cortical area due to sex (Table 6). Men did however, average greater metacarpal radiodensity and percent cortical area than women. This may be expected due to the generally larger bone mass of men compared to women. The mean TPI of the women was greater than that of the men. This may be indicative of a sex difference of a differential bone loss or original bone mass throughout the body.



Table 6. Trabecular pattern index (TPI), second metacarpal radiodensity and percent cortical area of men and women selected for radiological evaluation

	N	TPI	Second metacarpal	
			Radio density (mm Al)	Percent cortical area
Men	12	4.33	3.18	51.8
Standard deviation		±0.65	±0.31	±7.73
Women	27	4.52	2.89	46.8
Standard deviation		±0.80	±0.50	±11.52
F		0.19 <sub>NS</sub>	2.37 <sub>NS</sub>	4.11* <sub>.05</sub>

\* Although F was significant, the significance was due to selection. There was no significant difference due to sex.

Correlations of the radiological determinations of the subpopulation showed some interesting relationships. The correlation coefficient for right and left second metacarpal radiodensity was .72, and was .73 for the right and left second metacarpal percent cortical area. Correlating radiodensity and percent cortical area of each second metacarpal gave values of .61 for the left and .53 for the right hand. This indicates that bone quantity (as determined by percent cortical area) and quality (as determined by radiodensity) are changing in both hands but are progressing differently and perhaps independently. A negative relationship was found between age and TPI, indicating a general loss of trabecular bone with age. This is consistent with Morgan, et al. (37), Garn (17), and Western Regional findings (44) of a definite decrement in bone radiodensity with age.

Contrary to findings of Adams, et al. (1) no significant difference was found between right and left second metacarpal percent cortical

areas. Subsequently, right and left hand radiodensities and right and left hand percent cortical areas of the second metacarpal were combined for further analyses.

In Table 7, analysis of the subpopulation by the original criteria showed a significant difference in percent cortical area between the most-likely and least likely osteoporotic subjects. However, the greater cortical area was found in the most-likely osteoporotic group, as was the insignificant greater bone radiodensity. Trabecular pattern indices, however, were insignificantly greater in the least-likely osteoporotic group. Regardless of the designated group, bone radiodensity and percent cortical area in this instance appear to agree more closely with one another than either or both do with TPI. This may be due to the similarity of these parameters of different measurements of the same bone.

Table 7. Trabecular pattern index (TPI), second metacarpal bone radiodensity and percent cortical area of men and women selected as most-likely osteoporotic and least-likely osteoporotic by original selection criteria

	N	TPI	Second metacarpal	
			Radio-density (mm Al)	Percent cortical area
Most-likely osteoporotic	21	4.38	3.10	52.4
Standard deviation		$\pm 0.74$	$\pm 0.46$	$\pm 10.12$
Least-likely osteoporotic	18	4.56	2.85	43.6
Standard deviation		$\pm 0.92$	$\pm 0.45$	$\pm 9.04$
F		0.19 <sub>NS</sub>	2.37 <sub>NS</sub>	4.11*

\* Significant at 5 percent level

In Table 8, the same trend was also seen when TPI, radiodensity, and percent cortical area of women, selected according to the original criteria, were compared. Men subjects were not compared separately due to the small sample size of those considered least-likely osteoporotic (n=2).

Table 8. Trabecular pattern index (TPI), second metacarpal bone radiodensity and percent cortical area of women selected as most-likely osteoporotic and least-likely osteoporotic by original selection criteria

	N	TPI	Second metacarpal	
			Radio density (mm Al)	Percent cortical area
Most-likely osteoporotic	11	4.46	2.99	53.6
Standard deviation		$\pm 0.82$	$\pm 0.56$	$\pm 11.84$
Least-likely osteoporotic	16	4.56	2.83	42.1
Standard deviation		$\pm 0.96$	$\pm 0.47$	$\pm 8.46$
F		0.09 <sub>NS</sub>	0.65 <sub>NS</sub>	8.64*

\* Significant at 5 percent level

When the subpopulation was characterized by the radiological evaluations, analysis of variance showed a very significant difference in radiodensity and percent cortical area between osteoporotic and non-osteoporotic subjects, both sexes combined. Mean TPI was greater in osteoporotics than in non-osteoporotics, although insignificantly. In this case, measurements of the same bone agreed more closely with one another, but all indices were in agreement (Table 9).

Women were compared separately. Due to the small sample size of men radiologically classified, they were not compared separately.

Table 9. Trabecular pattern index (TPI), second metacarpal bone radiodensity and percent cortical area for radiologically determined osteoporotic and non-osteoporotic human subjects, women only

	Men and women combined				Women only			
	N	TPI	Second metacarpal		N	TPI	Second metacarpal	
			Radio- density (mm Al)	Percent cortical area			Radio- density (mm Al)	Percent cortical area
Osteoporotic	18	4.42	2.75	39.9	16	4.50	2.77	39.9
Standard deviation		±0.93	±0.51	± 9.87		±0.81	±0.48	±5.74
Non-osteoporotic	10	4.90	3.15	62.4	6	4.58	3.42	64.6
Standard deviation		±0.41	±0.29	± 4.63		±0.58	±0.33	±4.63
F		2.46 <sub>NS</sub>	3.78**	28.56**		0.05 <sub>NS</sub>	9.62**	88.93**

\*\* Significant at 1 percent level

Again, radiodensity and percent cortical area were significantly greater in non-osteoporotics than osteoporotics. Trabecular pattern indices were insignificantly greater.

In Table 10 the correlation coefficients for trabecular pattern index, radiodensity, and percent cortical area are shown for the selected subpopulation. The significant associations of these parameters seen in the two groups of men are attributable to the small sample size ( $n=2$ ).

The only significant correlations found were between radiodensity and percent cortical area. In general the correlations of these measurements were the most positive of the three correlations. This may be expected as these measurements were taken on the same bone. These correlations also indicate that bone changes in different parts of the body change independently of one another.

#### Discriminant function analysis

In an effort to manipulate the dietary and biochemical data to a greater potential for diagnosing osteoporosis, these data were processed in a discriminant function analysis to determine the success of specified criteria with respect to the radiological evaluations. The parameters of greatest potential significance used were estimated dietary protein and urinary calcium, total nitrogen and free alpha-amino nitrogen.

All data of each dietary or urinary component for all X-rayed individuals were combined to generate an "intercept" value,  $A_0$ , and a discriminant function constant,  $C$ . Coefficients for the dietary

Table 10. Correlation of trabecular pattern index (TPI), metacarpal radiodensity and percent cortical area (PCA) for osteoporotic and non-osteoporotic men and women by original selection criteria and radiological evaluation

	Correlation coefficient		
	TPI/Radiodensity	TPI/PCA	Radiodensity/PCA
<u>Original selection</u>			
(total)	-0.009	0.10	0.65
Osteoporotic			
Women	0.25	-0.01	0.83
Men	0.42	0.63	0.90
Non-osteoporotic			
Women	-0.18	0.16	0.37
	-1.00	1.00	-1.00
<u>Radiological evaluation</u>			
(total)	0.19	0.28	0.64
Osteoporotic			
Women	-0.07	0.23	0.27
Men	0.99	1.00	0.99
Non-osteoporotic			
Women	0.32	-0.07	0.72
Men	-0.60	-0.37	-0.50

and urinary values were generated respectively;  $A_1$ , dietary protein;  $A_2$ , total urinary nitrogen;  $A_3$ , urinary calcium;  $A_4$ , free alpha-amino nitrogen.

$$\begin{aligned} \text{The discriminant} &= A_0 + A_1 (\text{protein}) + A_2 (\text{total nitrogen}) \\ \text{function value/} & \\ \text{individual} & \\ & A_3 (\text{calcium}) + A_4 (\text{free alpha-amino} \\ & \quad \text{nitrogen}) \end{aligned}$$

where an individual's values for protein, nitrogen, etc. are used. Groups of origin and classification are established. Group 1 for radiological origin is non-osteoporotic, Group 2 is osteoporotic. If the discriminant function value was greater than C, membership for that individual is classified as Group 1, non-osteoporotic, regardless of the radiological origin. If the discriminant function value was less than C, classification for that individual is Group 2, osteoporotic. The rate of success is determined by the number of successful classifications of the same origin.

		Discriminant function classification	
		1	2
Radiological origin	1	$X_{1,1}$	$Y_{1,2}$
	2	$X_{2,1}$	$Y_{2,2}$

$$\text{Rate of success} = \frac{X_{1,1} + Y_{2,2}}{X_{1,1} + X_{2,1} + Y_{1,2} + Y_{2,2}}$$

Figure 1. Determination of rate of success of discriminant function analysis

The rate of success of the discriminant function analysis in this study in the October sampling was 0.73; in March, 0.81; and combined, 0.68. Other combinations of parameters have not been tested. By varying the dietary and urinary parameters, the rate of success may be improved.

The selection of the subpopulation for this study could not be selected with the benefit of discriminant function testing. However, by varying the variables used in the function determinant, better rates of success may be determined within a given group and projected for other populations.



## SUMMARY

Through computer analysis of dietary records and biochemical analysis of urine samples, this study has shown seasonal variations in estimated dietary consumption of selected nutrients, and very significant seasonal changes in urinary excretions unaccounted for by dietary increases.

Evaluation of radiological exposures in terms of metacarpal radiodensity and percent cortical area and trabecular pattern index showed significant differences in metacarpal measurements between radiologically evaluated osteoporotic and non-osteoporotic subjects. Percent cortical area was consistently the most sensitive measurement in agreement with the total radiological evaluation. This is especially significant in supporting the description of osteoporosis as a decrease of bone quantity but not of bone quality. The trabecular pattern index did not at any time correlate with the metacarpal measurements. The failure of the trabecular pattern index to coincide with the metacarpal measurements does not diminish its value in determining osteoporosis in trabecular bone, but rather further indicates that osteoporosis differentially affects bones in varying parts of the body.

The discrepancy of the groups considered most- and least-likely osteoporotic by the original selection criteria of dietary and urinary analyses alone and the radiologically confirmed groups indicates that as yet osteoporosis is not yet determinable by these parameters.

### Recommendations

As the diet at the time of selection of osteoporosis study may not be indicative of the diet during the development of the condition, emphasis on diagnosing osteoporosis from outside parameters should be placed on urine analyses from extended monitoring. The potential of the efficacy of the discriminant function analysis should be pursued by using urinary analyses from individuals who have already been radiologically evaluated as osteoporotic; manipulated to determine which variables may be best used for consistently best results.

## LITERATURE CITED

1. Adams, P., G. T. Davies, and P. M. Sweetnam. 1969. Observer error and measurements of the metacarpal. *British Journal of Radiology* 42:192-197.
2. Albright, F., P. H. Smith, and A. M. Richardson. 1941. Post-menopausal osteoporosis, its clinical features. *Journal of the American Medical Association* 116:2465-2474.
3. Association of Official Analytical Chemists. 1975. *Official Methods of Analysis of the Association of Official Analytical Chemists*. Association of Official Analytical Chemists, Washington, D. C. 12th ed. 1094 p.
4. Bayless, T. M., and N. S. Toseweig. 1966. A racial difference in incidence of lactase deficiency. *Journal of the American Medical Association* 197:968-972.
5. Bernstein, D. S., N. Sadowsky, D. M. Hegsted, C. D. Guri, and F. J. Stare. 1966. Prevalence of osteoporosis in high-fluoride and low-fluoride areas in North Dakota. *Journal of the American Medical Association* 198:400-504.
6. Birge, S. J., Jr., H. T. Keutmann, P. Cuatrecasas, and G. D. Whedon. 1967. Osteoporosis, intestinal lactase deficiency and low dietary calcium intake. *New England Journal of Medicine* 276:446-448.
7. Brink, M. F., E. W. Speckmann, and M. Balsley. 1968. Current concepts in geriatric nutrition. *Geriatrics* 23:113-120.
8. Buhr, A. J., and A. M. Cooke. 1959. Fracture patterns. *Lancet* i:531.
9. Chu, J-Y., S. Margen, and F. M. Costa. 1975. Studies in calcium metabolism. II Effects of low calcium and variable protein intake on human calcium metabolism. *American Journal of Clinical Nutrition* 28:1028-1035.
10. Cohn, S. H., C. S. Donbrowski, W. Hauser, and H. L. Atkins. 1968. High calcium diet and the parameters of calcium metabolism in osteoporosis. *American Journal of Clinical Nutrition* 21:1246-1253.
11. Dallas, I., and B. E. C. Nordin. 1962. The relation between calcium intake and roentgenologic osteoporosis. *American Journal of Clinical Nutrition* 11:263-269.

12. Davis, M. E., N. M. Strandford, and L. H. Lanzl. 1966. Estrogens and the aging process: the detection, prevention, and retardation of osteoporosis. *Journal of the American Medical Association* 196:219-224.
13. Epker, B. N. 1968. Bone remodeling and balance and the development of osteoporosis. *Parodontologie and Academy Review* 2:125-135.
14. Epker, B. N., and H. M. Frost. 1965. A histological study of remodeling at the periosteal, haversian canal, cortical endosteal, and trabecular periosteal surfaces in human rib. *Anatomical Record* 152:129-136.
15. Fourman, P., R. Roger, M. J. Levell, and D. B. Morgan. 1968. *Calcium Metabolism and the Bone*. Blackwell Scientific Publications, Oxford and Edinburgh. 2nd ed. 656 p.
16. Garn, S. M. 1967. Nutrition and bone loss. Introductory remarks. *Federation Proceedings; Federation of American Societies for Experimental Biology* 26:1716.
17. Garn, S. M. 1972. The course of bone gain and the phases of bone loss. *Orthopedic Clinics of North America* 3:503-520.
18. Garn, S. M. 1975. Bone loss and aging. In *Physiology and Pathology of Human Aging*. Academic Press, Inc., San Francisco, pp. 39-57.
19. Garn, S. M. and A. K. Poznanski. 1973. The simultaneous radiogrammetric approach to the two bone surfaces. In *Clinical Aspects of Metabolic Bone Disease*. B. Frame, A. M. Parfitt, and H. Duncan (Eds.). *Excerpta Medica Amsterdam* pp. 20-24.
20. Garn, S. M., A. K. Poznanski, and J. M. Nagy. 1971. Bone measurements in the differential diagnosis of osteopenia and osteoporosis. *Radiology* 100(3):509-518.
21. Garn, S. M., C. G. Rohmann, and B. Wagner. 1967. Bone loss as a general phenomenon in man. *Federation Proceedings; Federation of American Societies for Experimental Biology* 26:1729-1736.
22. Gomori, G. 1953. Calcium, magnesium, and phosphorus in biological material. In *Standard Methods of Clinical Chemistry*, vol. 1, Academic Press, New York.
23. Harrison, R., R. Fraser, and B. Mullan. 1961. Calcium metabolism in osteoporosis. *Lancet* 1:1015.
24. Heany, R. P. 1965. A unified concept of osteoporosis. *American Journal of Medicine* 39:877-880.

25. Hegsted, D. M. 1967. Mineral intake and bone loss. Federation Proceedings; Federation of American Societies for Experimental Biology 26:1747-1754.
26. Houssain, J., D. A. Smith, and B. E. C. Nordin. 1970. Parathyroid activity and postmenopausal osteoporosis. Lancet 1:809.
27. Hurthal, S. M., and G. P. Vose. 1969. The relationship of dietary calcium intake to radiographic bone density and normal and osteoporotic persons. Calcified Tissue Research 4:245-255.
28. Israel, H. 1967. Loss of bone and remodeling-redistribution in the craniofacial skeleton with age. Federation Proceedings; Federation of American Societies for Experimental Biology 26:1723-1728.
29. Jowsey, J. and K. A. Johnson. 1972. Juvenile osteoporosis: Bone findings in seven patients. Journal of Pediatrics 81(3): 511-517.
30. Lafferty, F. W., G. E. Spencer, Jr., and O. H. Pearson. 1964. Effects of androgens, estrogens and high calcium intakes on bone formation and resorption in osteoporosis. American Journal of Medicine 36:514-528.
31. Linkswiler, H. S., C. L. Joyce, and C. R. Anand. 1974. Calcium retention of young adult males as affected by level of protein and of calcium intake. New York Academy of Sciences, Series II, 36(4):333-340.
32. Lorentz, K., and B. Flatter. 1974. Simplified colorimetry of alpha-amino nitrogen in plasma, serum, or urine. Clinical Chemistry 20:1553-1555.
33. Lutwak, L. 1974. Continuing need for dietary calcium throughout life. Geriatrics 29:171-174.
34. Meema, H. E. 1963. Cortical bone atrophy and osteoporosis as a manifestation of aging. American Journal of Roentgenology 89:1287-1295.
35. Meema, H. E., and S. Meema. 1974. Involutional (physiologic) bone loss in women and the feasibility of preventing structural failure. Journal of the American Geriatric Society 22(10): 443-452.
36. Morgan, B. 1973. Osteomalacia, Renal Osteodystrophy, and Osteoporosis. I. Newton Kugelmass (Ed.), Charles Thomas Publisher, Springfield, Illinois. 423 p.
37. Morgan, D. B., C. N. Pulvertaft, and P. Fourman. 1966. Effects of age on the loss of bone after gastric surgery. Lancet 2:772.

38. Morgan, D. B., F. W. Spiers, C. N. Pulvertaft, and P. Fourman. 1967. The amount of bone in the metacarpal and the phalanx according to age and sex. *Clinical Radiology* 18:101-108.
39. Newton-John, J. F., and D. B. Morgan. 1968. Osteoporosis: Disease or senescence? *Lancet* 1:232-233.
40. Nordin, B. E. C. 1962. Calcium balance and calcium requirement in psinal osteoporosis. *Americal Journal of Clinical Nutrition* 10:384-390.
42. Nordin, B. E. C. 1966. International patterns of osteoporosis. *Clinical Orthopaedics and Related Research* 45:17-30.
43. Nordin, B. E. C., J. Aaron. J. C. Gallagher, and A. Horsman. 1972. Calcium and bone metabolism in old age. In *Nutrition in Old Age. Symposia of the Swedish Nutrition Foundation, the Swedish Nutrition Foundation.* Almquist and Wiksell, Uppsala. 180 p.
44. Oland, L. M., K. P. Warnick, and N. C. Esselbough. 1958. Cooperative nutritional status studies in the Western Region. II. Bone Density. *Montana Agricultural Experiment Bulletin* 534.
45. Popowitz, M., and A. D. Johnston. 1971. Osteoporosis: Controlled methods of measurement. *Clinical Orthopaedics and Related Research* 74:185-195.
46. Posner, A. 1967. Relationship between diet and bone mineral ultrastructure. *Federation Proceedings; Federation of American Societies for Experimental Biology* 26:1717-1722.
47. Rich, C., J. Ensink, and P. Ivanovich. 1964. The effects of sodium fluoride on calcium metabolism of subjects with metabolic bone disease. *Journal of Clinical Investigation* 43:545-555.
48. Smith, D. M., M. R. A. Khairi, and C. Johnston, Jr. 1975. The loss of bone mineral with aging and its relationship to risk of fracture. *Journal of Clinical Investigation* 56:311-318.
49. Smith, R. W. 1967. Dietary and hormonal factors in bone loss. *Federation Proceedings; Federation of American Societies for Experimental Biology* 26:1734-1746.
50. Smith, R. W., and J. Rizek. 1966. Epidemiological studies of osteoporosis in women of Puerto Rico and Southeastern Michigan with special reference to age, race, national origin and to other related ot associated findings. *Clinical Orthopaedics and Related Research* 45:31-48.
51. Spencer, H., J. Menczel, I. Lewin, and J. Samachson. 1964. Absorption of calcium in osteoporosis. *American Journal of Medicine* 37:223-234.

52. Stein, I., and M. L. Beller. 1970. Therapeutic progress in osteoporosis. *Geriatrics* 25:159-163.
53. Thin, C. G., and P. A. Thompson. 1967. Estimation of calcium and magnesium in serum and urine by atomic absorption spectrophotometry. *Journal of Clinical Pathology* 20:280-282.
54. Vinther-Paulsen, N. 1953. Calcium and phosphorus intake in senile osteoporosis. *Geriatrics* 8:76-79.
55. Walker, A. R. P. 1965. Osteoporosis and calcium deficiency. *American Journal of Clinical Nutrition* 16:327-328.
56. Watt, B. K., and A. L. Merrill. 1963. Composition of Foods, Agricultural Handbook No. 8. Agricultural Research Service, United States Department of Agriculture, Washington, D. C. 190 p.

## APPENDICES



Appendix AIndividual Information Sheets

## INFORMED CONSENT AGREEMENT

UTAH STATE UNIVERSITY

Project Title: Senior's Nutrition Aide Program: An  
Evaluation of the Five-County Action  
Program

I hereby give my consent to participate in the above named project involving human subjects. I understand the procedure to be followed in the study and am aware of the discomforts and risks involved by my participation. I will receive answers to any inquiries regarding the project and am free to withdraw my consent and discontinue participation in the project at any time.

---

signed

date

Name \_\_\_\_\_

## DIETARY INTAKE RECORD

Are there any foods you cannot eat? For instance, are you a diabetic or do you have any food allergies? Please list.

\_\_\_\_\_  
\_\_\_\_\_

Has your doctor put you on a special diet? Check which ones:

Bland \_\_\_\_\_ Salt Restricted \_\_\_\_\_

Fat Resistant \_\_\_\_\_ Weight Reduction \_\_\_\_\_

Other (tell which kind): \_\_\_\_\_

Are you taking any vitamin pills or mineral pills? Yes \_\_\_\_\_ No \_\_\_\_\_

If yes, did a doctor recommend taking them? Yes \_\_\_\_\_ No \_\_\_\_\_

Please list the vitamins or minerals contained in the pill:

\_\_\_\_\_  
\_\_\_\_\_

PLEASE LIST ALL FOODS AND DRINKS YOU CONSUME DURING THE DAY

Approximate time you ate the food	What kind of food did you eat? Be specific.	How much of each food did you eat?	How was the food prepared? Boiled, fried, raw, toasted, etc.
Breakfast			
Snack			
Lunch			
Snack			
Dinner			
Snack			

NUMBER \_\_\_\_\_

NAME \_\_\_\_\_

AGE \_\_\_\_\_

SEX \_\_\_\_\_

HEIGHT \_\_\_\_\_

WEIGHT \_\_\_\_\_

BLOOD PRESSURE \_\_\_\_\_

URINE VOLUME \_\_\_\_\_

URINE SUGAR \_\_\_\_\_

URINE SPECIFIC GRAVITY \_\_\_\_\_

HEMOGLOBIN \_\_\_\_\_

HEMATOCRIT \_\_\_\_\_

## INFORMED CONSENT AGREEMENT

## UTAH STATE UNIVERSITY

I hereby give my consent to participate in the project involving X-ray procedures additionally involved with the SNAP nutrition evaluation, and am aware of the discomforts and risks involved by my participation. I will receive answers to any inquiries regarding the project and am free to withdraw my consent and discontinue participation in the project at any time for any reason.

---

signed

date

Appendix BComputer Statistical Analyses

Table 11. Means and standard deviations for diet and urine variables, October

VARIABLE	MEAN	STANDARD DEVIATION
SP GR 1	1.01390	0.00499
U VOL 2	1338.58182	528.10929
D CA 3	633.60000	289.65272
D P 4	985.20000	312.92968
D PROT 5	58.70000	17.59459
D B6 6	1.03818	0.45251
BT N 7	5.13105	3.05665
B CA 8	54.86364	79.18524
B P 9	485.86364	297.45673
FREE N 10	94.83636	63.26245
P BAL 11	499.33636	417.23982
UC/DC 12	0.11304	0.18615
DC/DP 13	0.63349	0.17740
DC/P 14	11.22017	5.44000
BC/TN 15	12.39407	14.44589



Table 12. Correlation matrices for variables, October

CORRELATION MATRIX

VARIABLE NUMBER	1	2	3	4	5	6	7	8	9	10
1	1.000									
2		1.000								
3			1.000							
4				1.000						
5					1.000					
6						1.000				
7							1.000			
8								1.000		
9									1.000	
10										1.000

Table 13. Means and standard deviations for diet and urine variables, March

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VARIABLE		MEAN	STANDARD DEVIATION
SP GR	1	1.01505	0.00418
U VOL	2	1315.28829	624.71657
D CA	3	726.52252	324.28817
D P	4	1042.98198	332.78158
O PROT	5	63.69369	16.00670
D 86	6	1.09099	0.45378
BT N	7	8.69825	5.83913
B CA	8	99.44144	117.93617
B P	9	1080.63964	755.51970
FREE N	10	214.27928	122.93163
P BAL	11	-37.65766	765.33691
UC/DC	12	0.16442	0.20377
DC/DP	13	0.68461	0.20492
DC/P	14	11.25520	3.79834
BC/TN	15	13.01502	15.55796

Table 14. Correlation matrices for variables, March

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VARIABLE	MEAN	STANDARD DEVIATION
SP GR 1	1.01448	0.00462
U VOL 2	1326.88235	577.45445
D CA 3	680.27149	310.35112
D P 4	1014.22172	323.61650
D PROT 5	61.20814	16.96312
D B6 6	1.06471	0.45289
BT N 7	6.92272	4.98723
B CA 8	77.25339	102.76265
B P 9	784.59729	646.59329
FREE N 10	154.82805	114.55026
P BAL 11	229.62443	671.96753
UC/DC 12	0.13885	0.19645
DC/DP 13	0.65917	0.19299
DC/P 14	11.23777	4.67721
BC/TN 15	12.70595	14.98383

---

Table 15. Means and standard deviations for diet and urine variables, combined

CORRELATION MATRIX

VARIABLE NUMBER	1	2	3	4	5	6	7	8	9	10
1	1.000									
2		1.000								
3			1.000							
4				1.000						
5					1.000					
6						1.000				
7							1.000			
8								1.000		
9									1.000	
10										1.000

Table 16. Correlation matrices for variables, combined

CORRELATION MATRIX

VARIABLE NUMBER	1	2	3	4	5	6	7	8	9	10
1	1.000									
2		1.000								
3			1.000							
4				1.000						
5					1.000					
6						1.000				
7							1.000			
8								1.000		
9									1.000	
10										1.000

Appendix CFrequency Distribution of Dietary and Urinary Variablesby Sex and Season

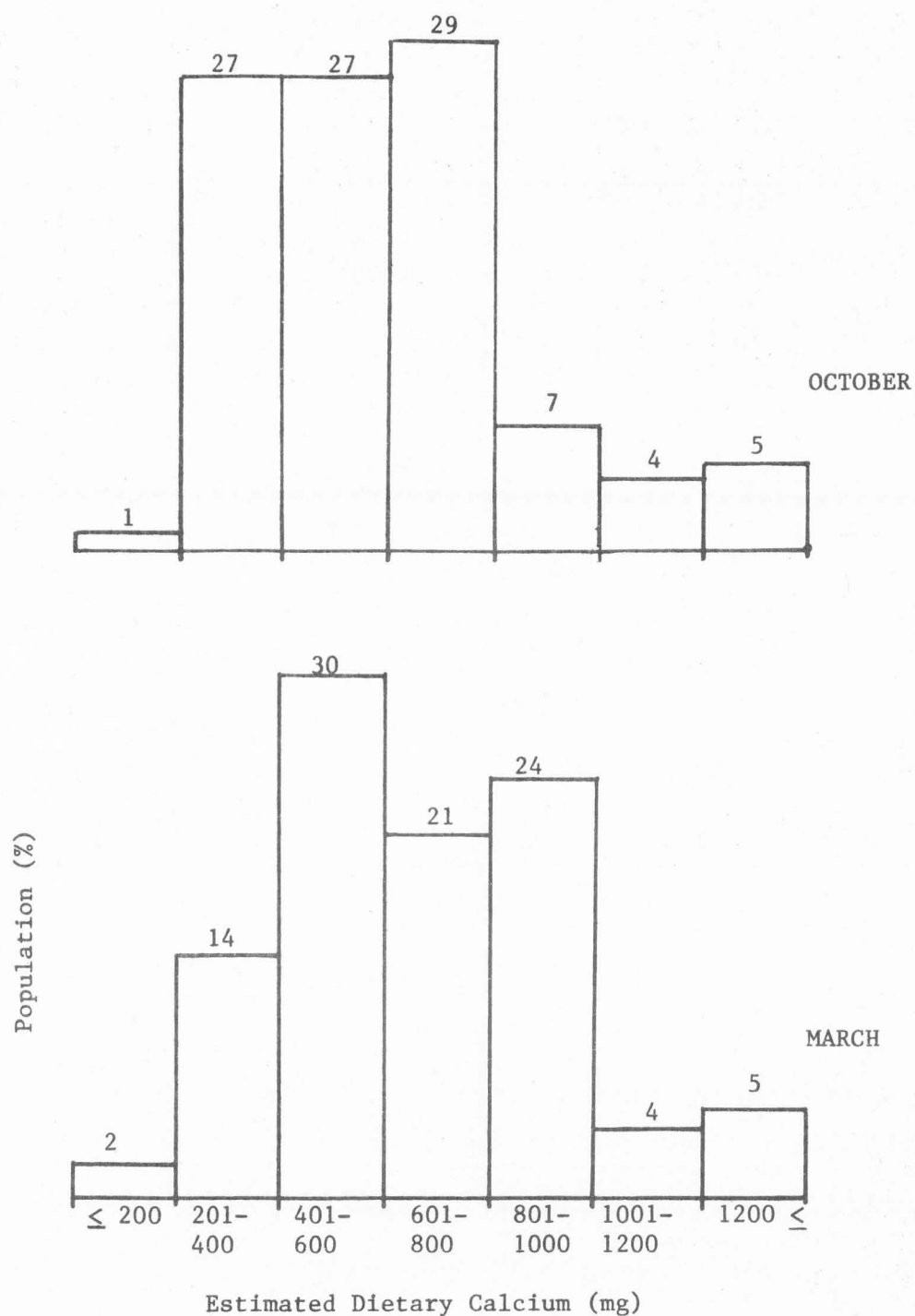


Figure 2. Frequency distribution of estimated dietary calcium intake for women, October and March.

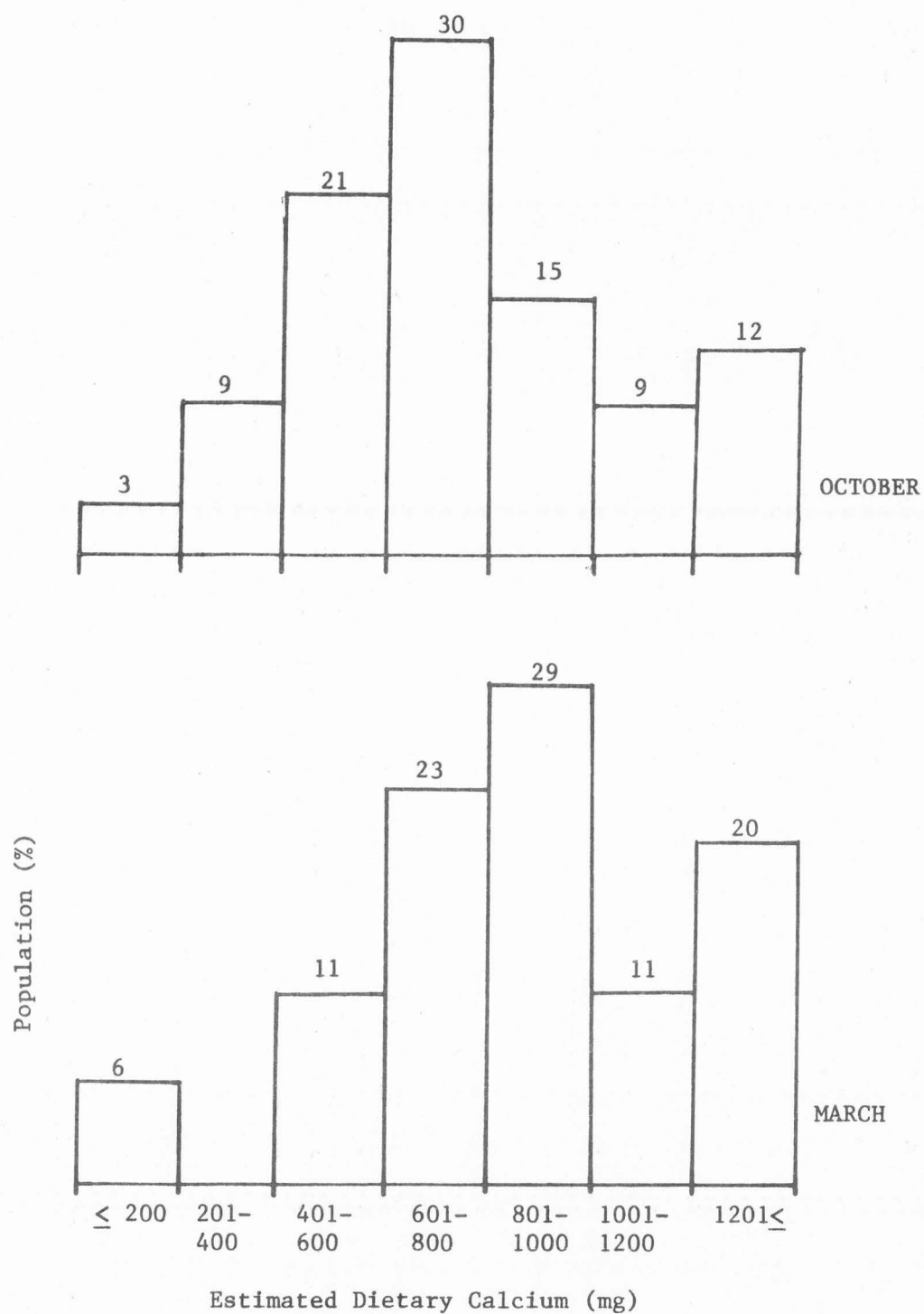


Figure 3. Frequency distribution of estimated dietary calcium intake for men, October and March.



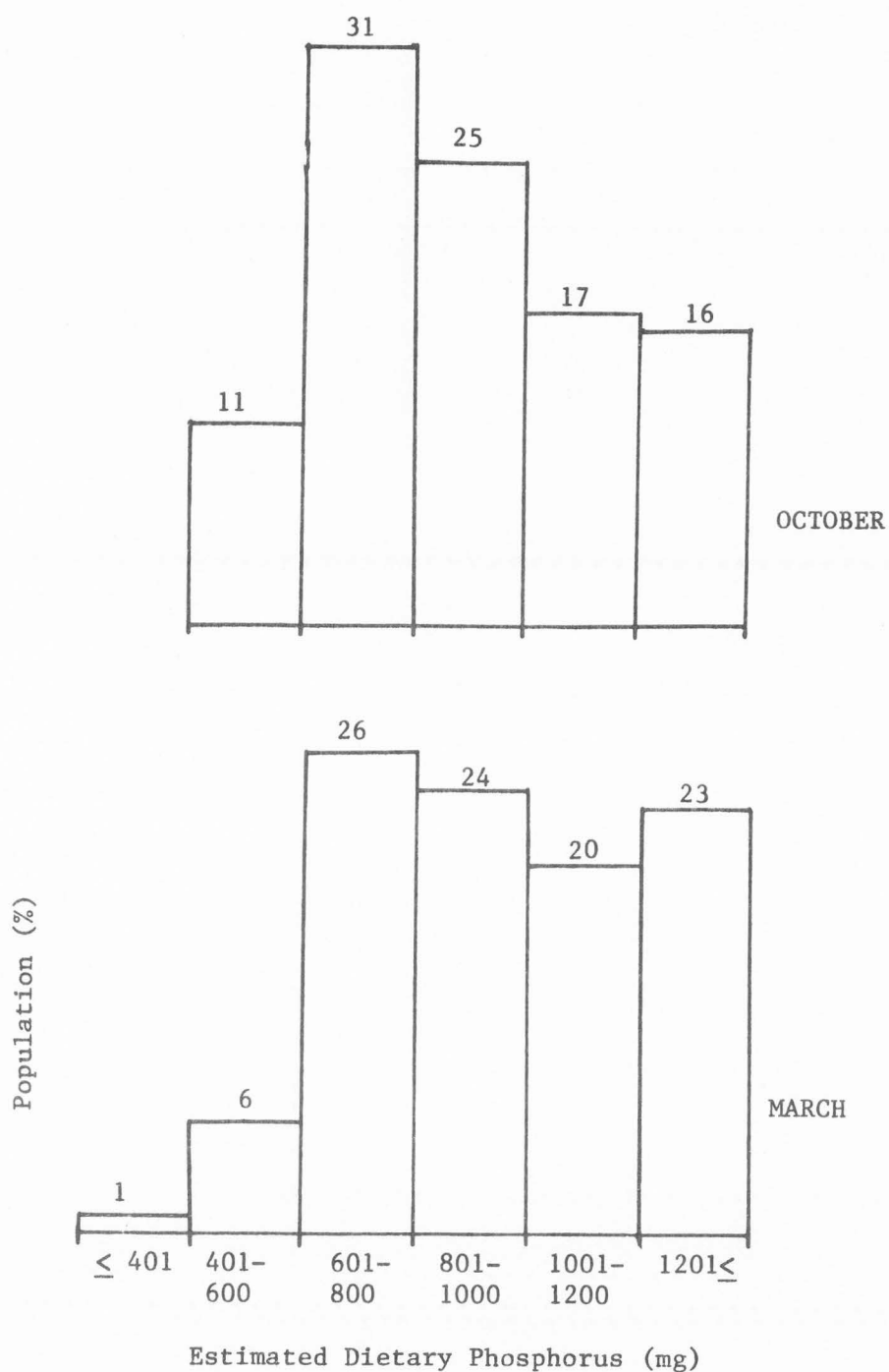


Figure 4. Frequency distribution of estimated dietary phosphorus intake for women, October and March.

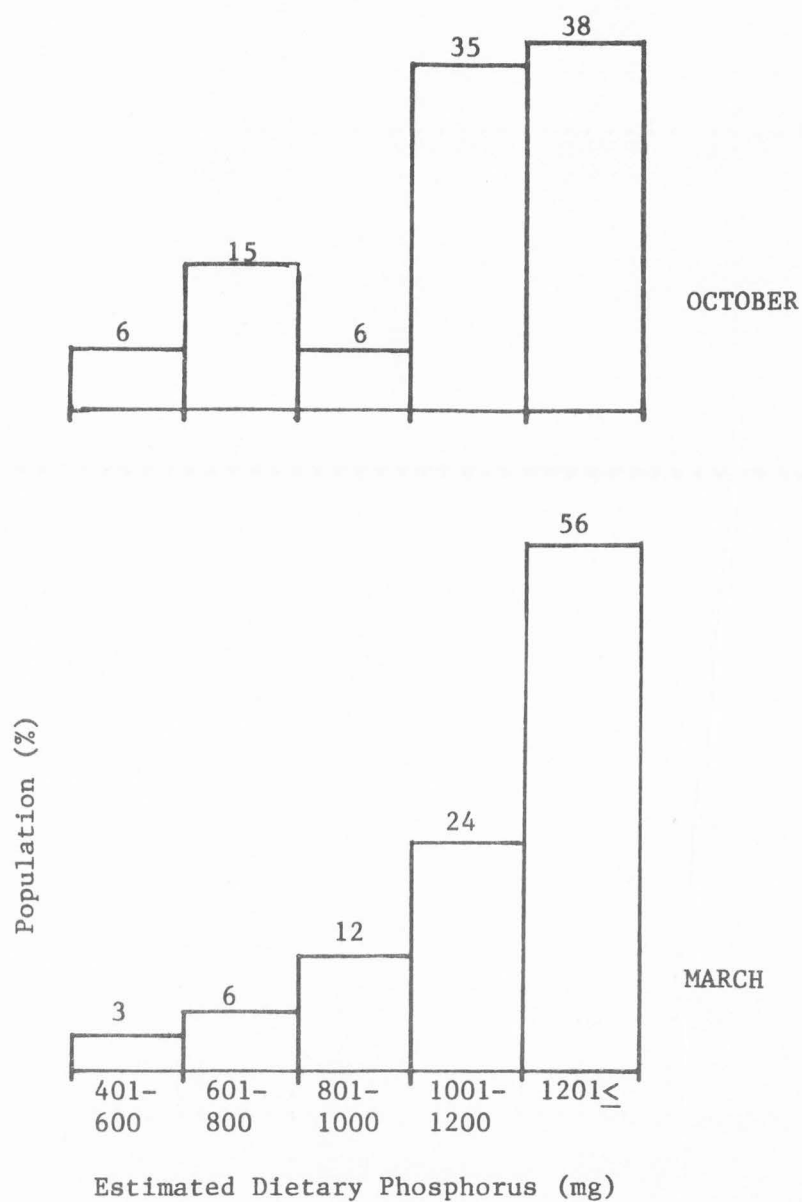


Figure 5. Frequency distribution of estimated dietary phosphorus intake for men, October and March.

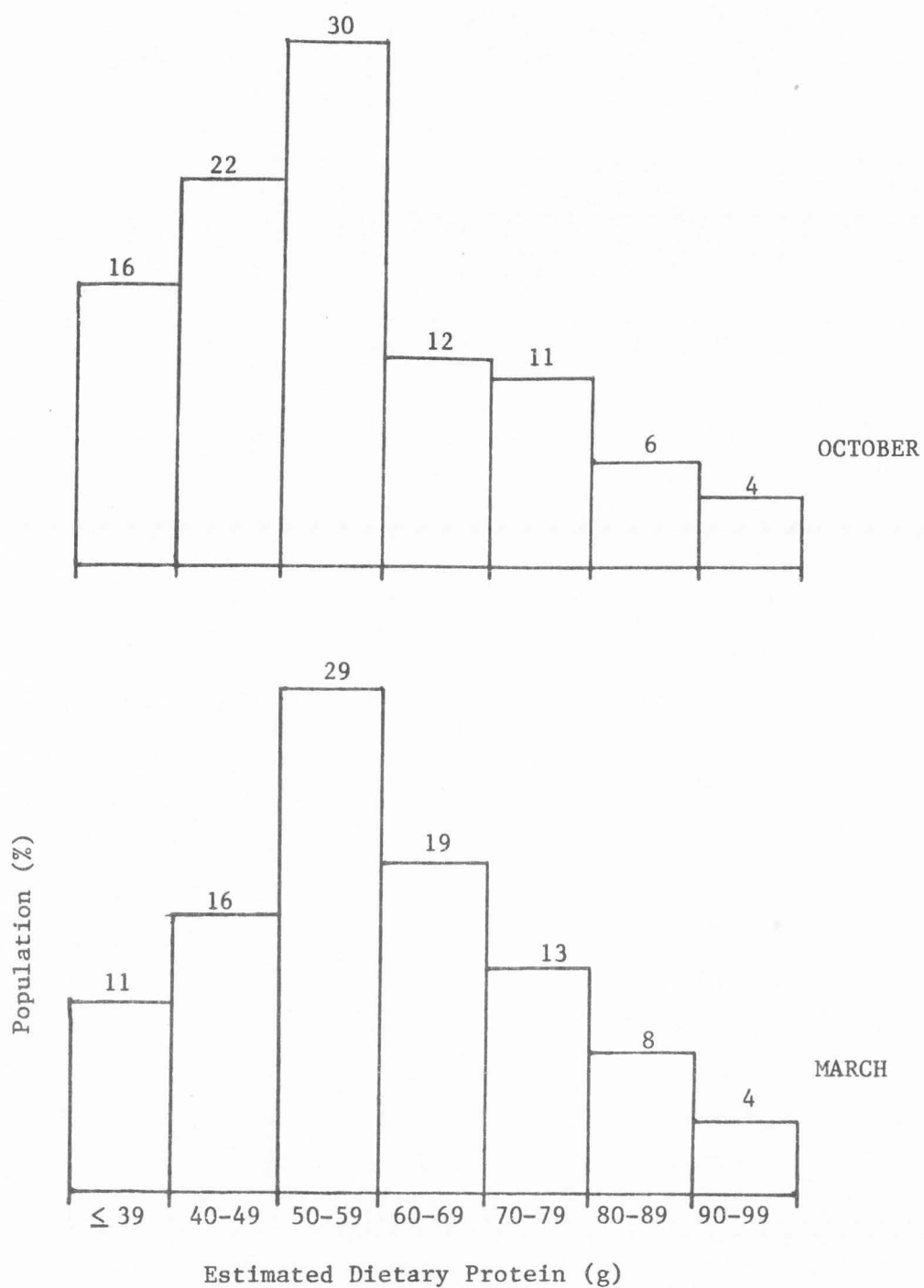


Figure 6. Frequency distribution of estimated dietary protein intake for women, October and March.

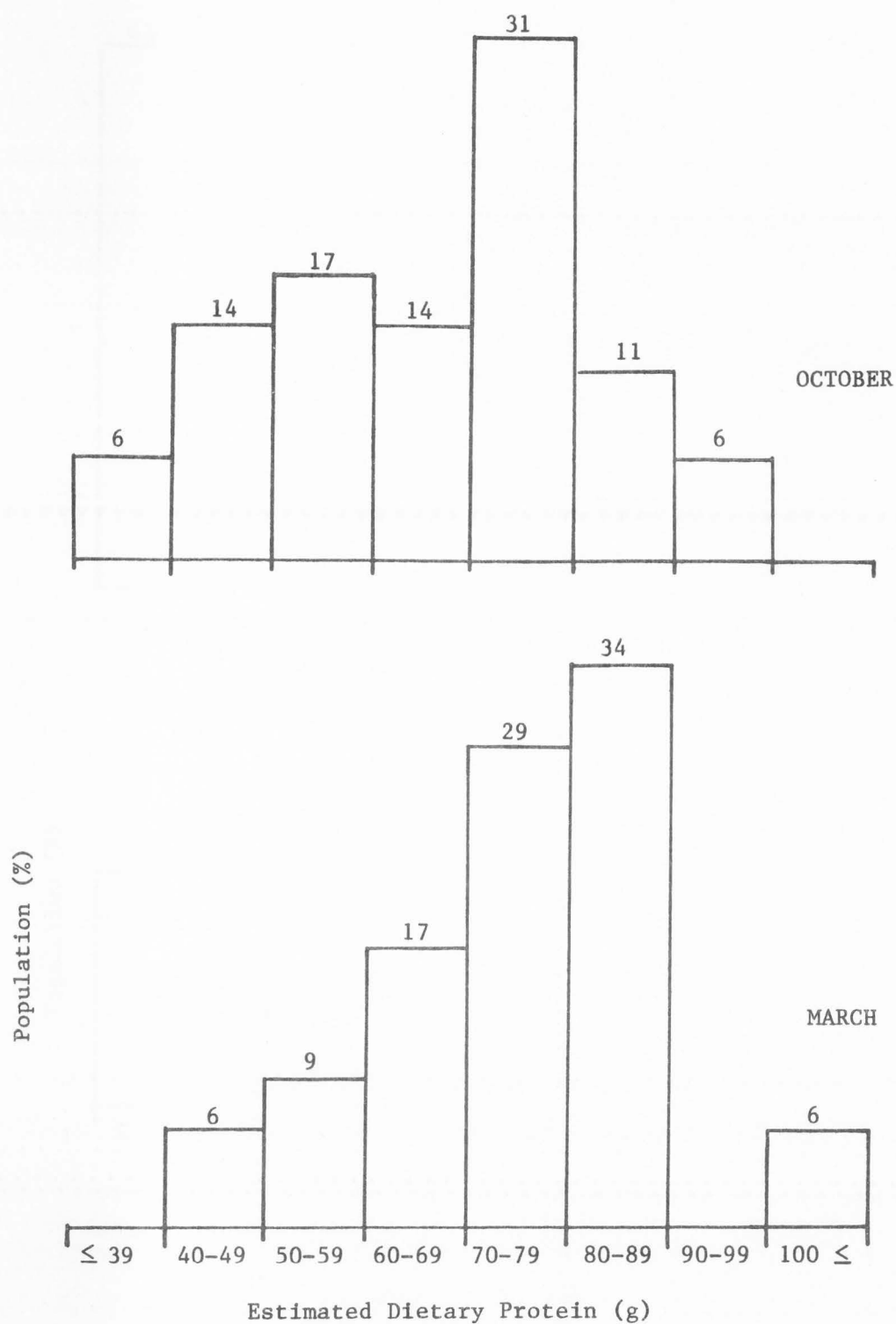


Figure 7. Frequency distribution of estimated dietary protein intake for men, October and March.

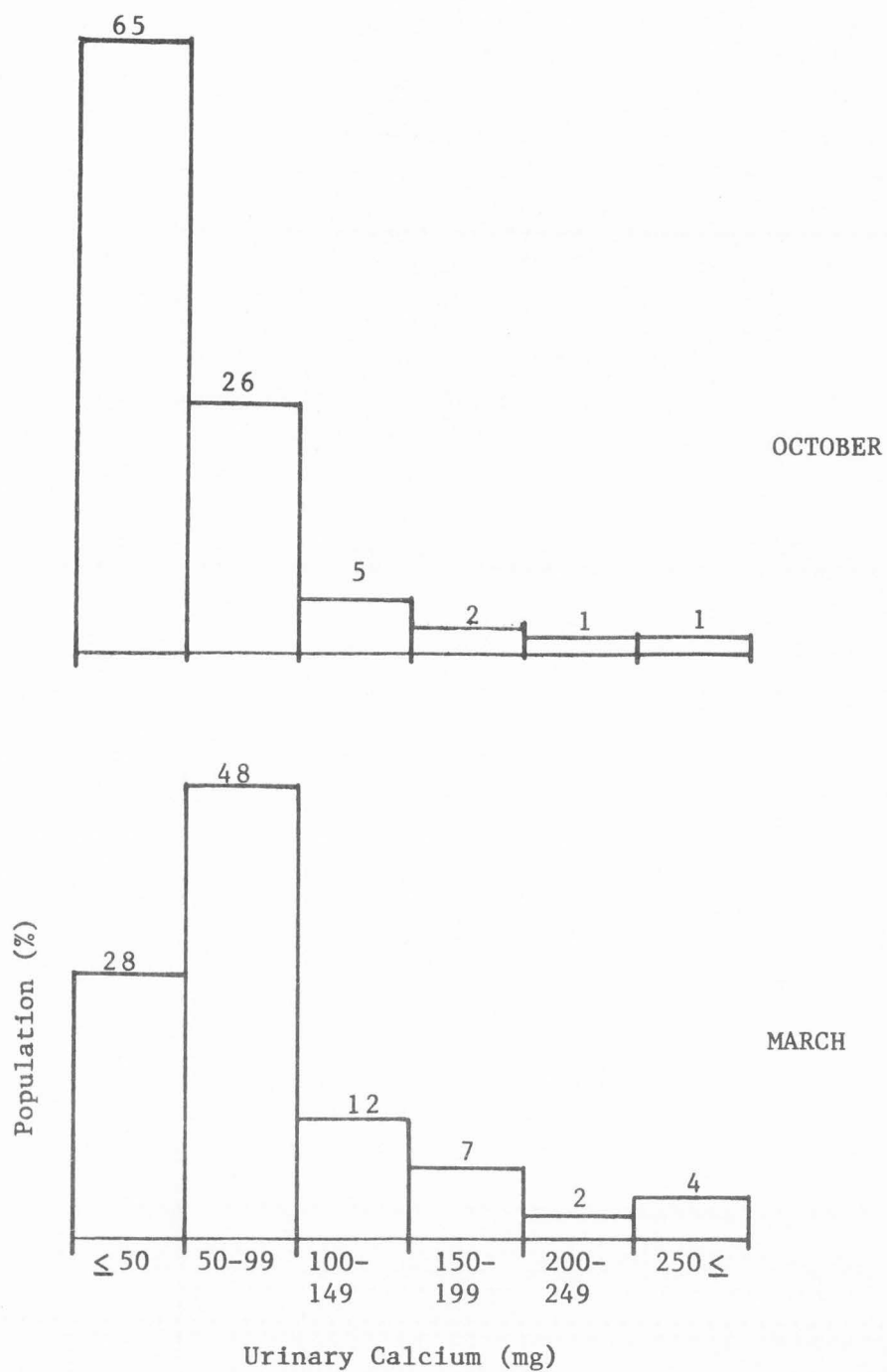


Figure 8. Frequency distribution of urinary calcium excretion for women, October and March.

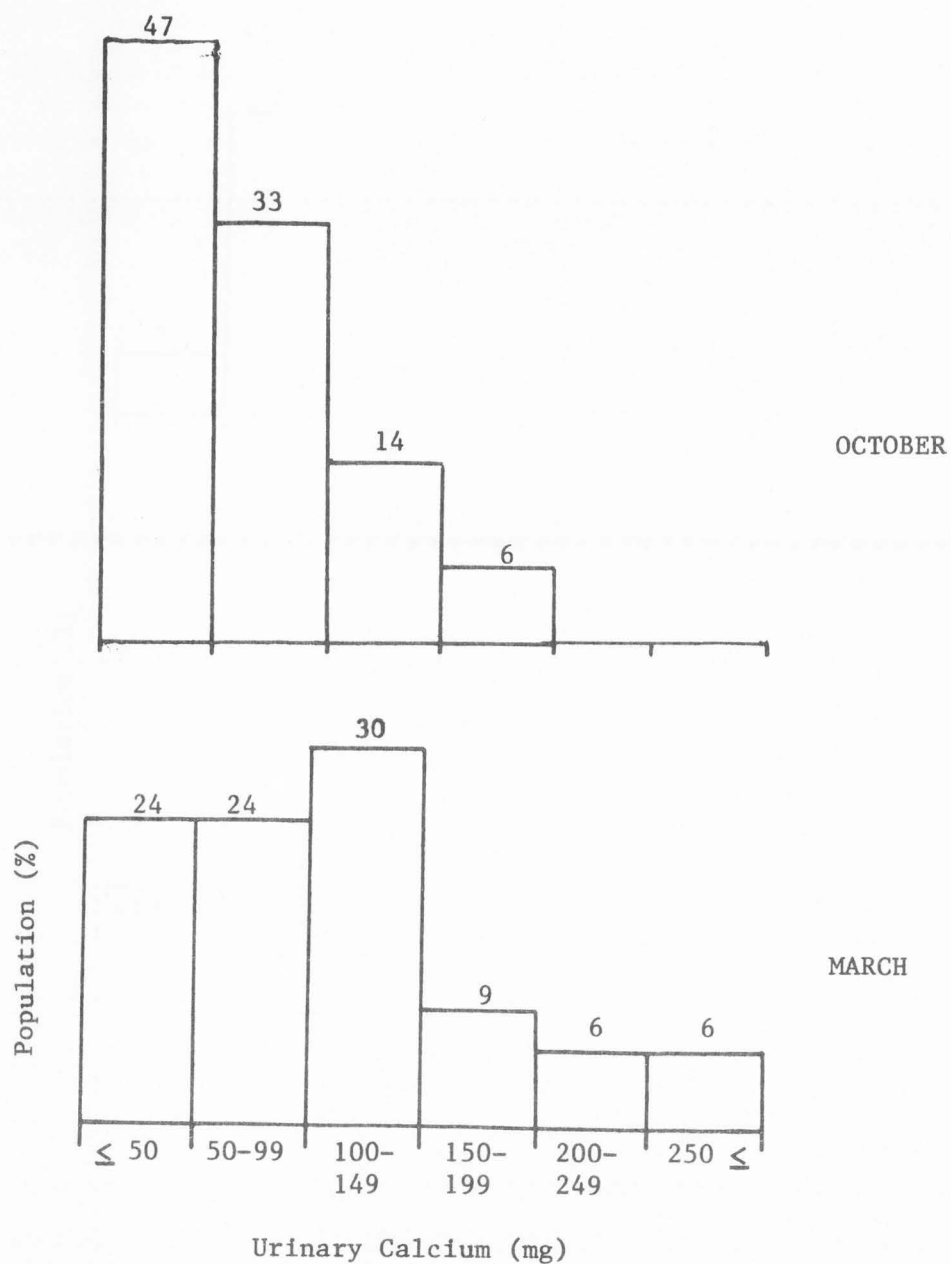


Figure 9. Frequency distribution of urinary calcium excretion for men, October and March.

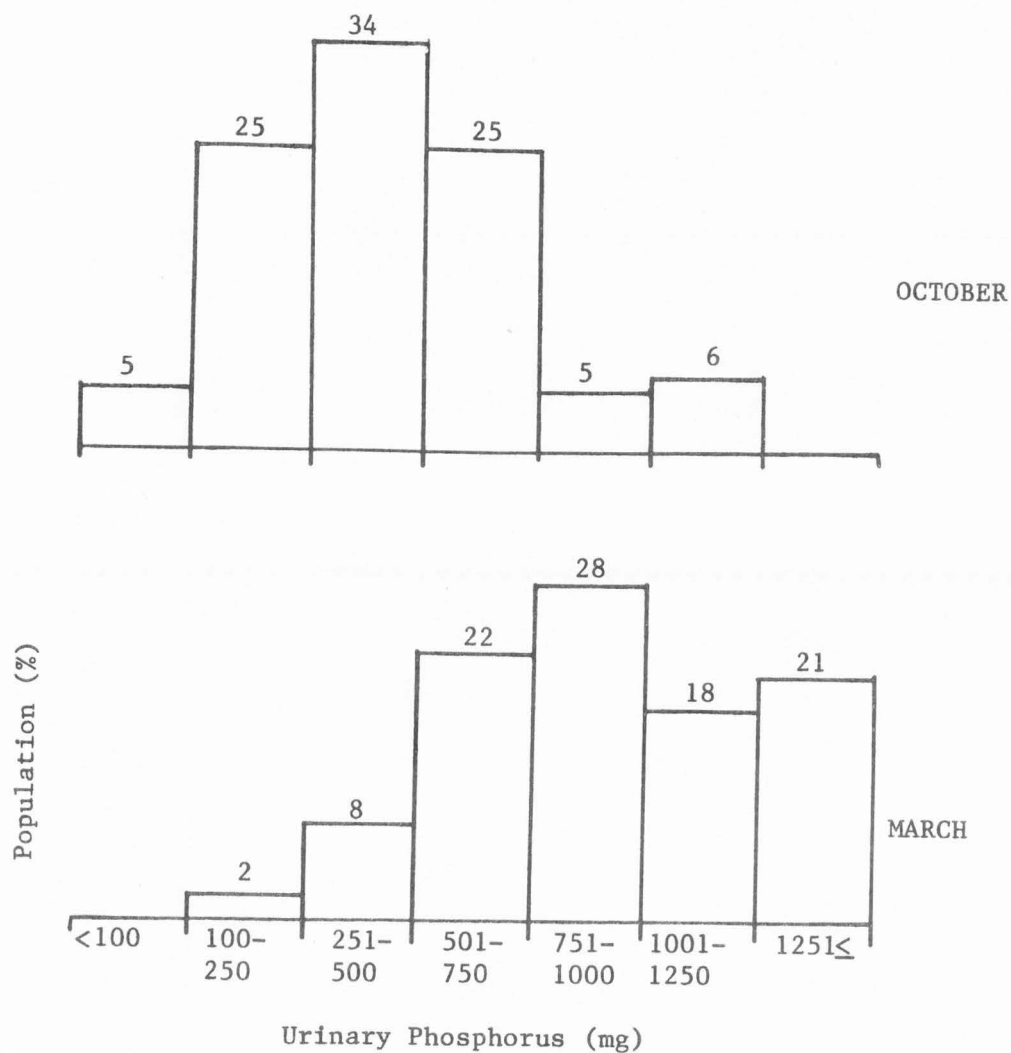


Figure 10. Frequency distribution of urinary phosphorus excretion for women, October and March.

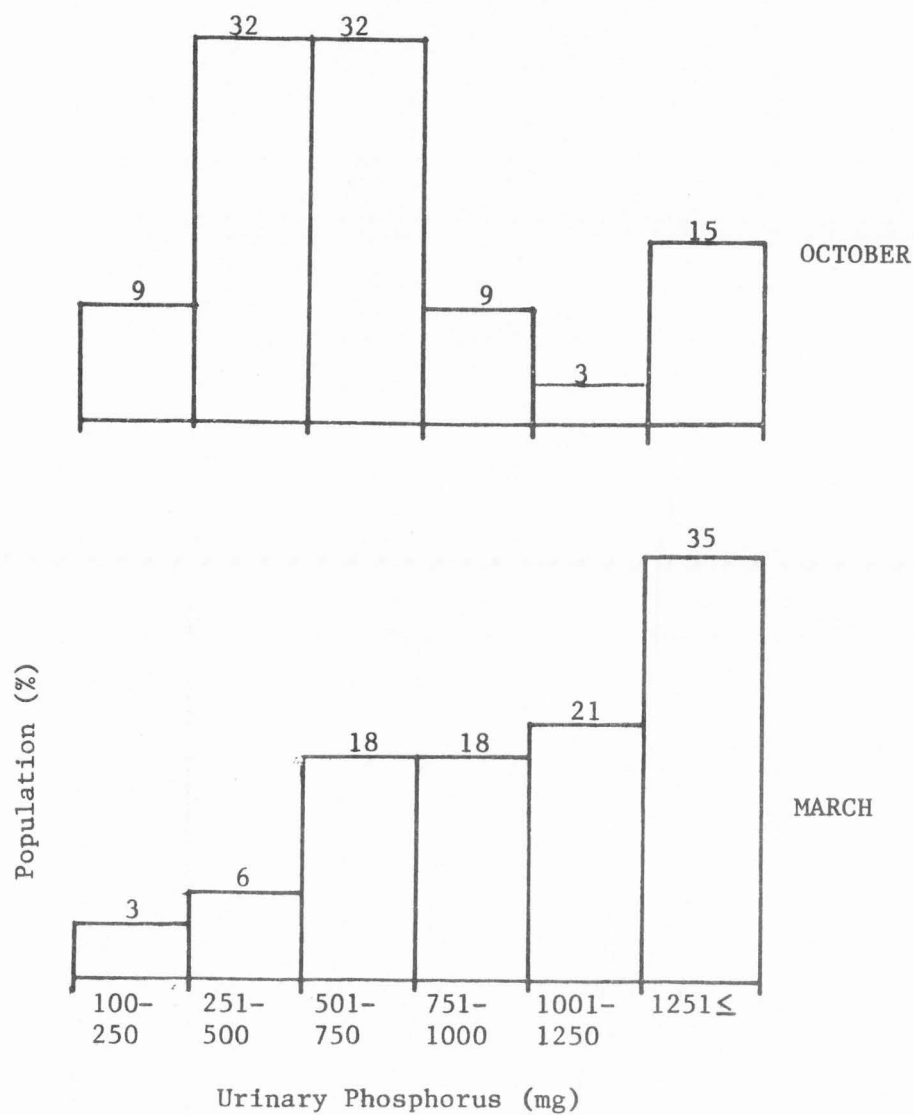


Figure 11. Frequency distribution of urinary phosphorus excretion for men, October and March.



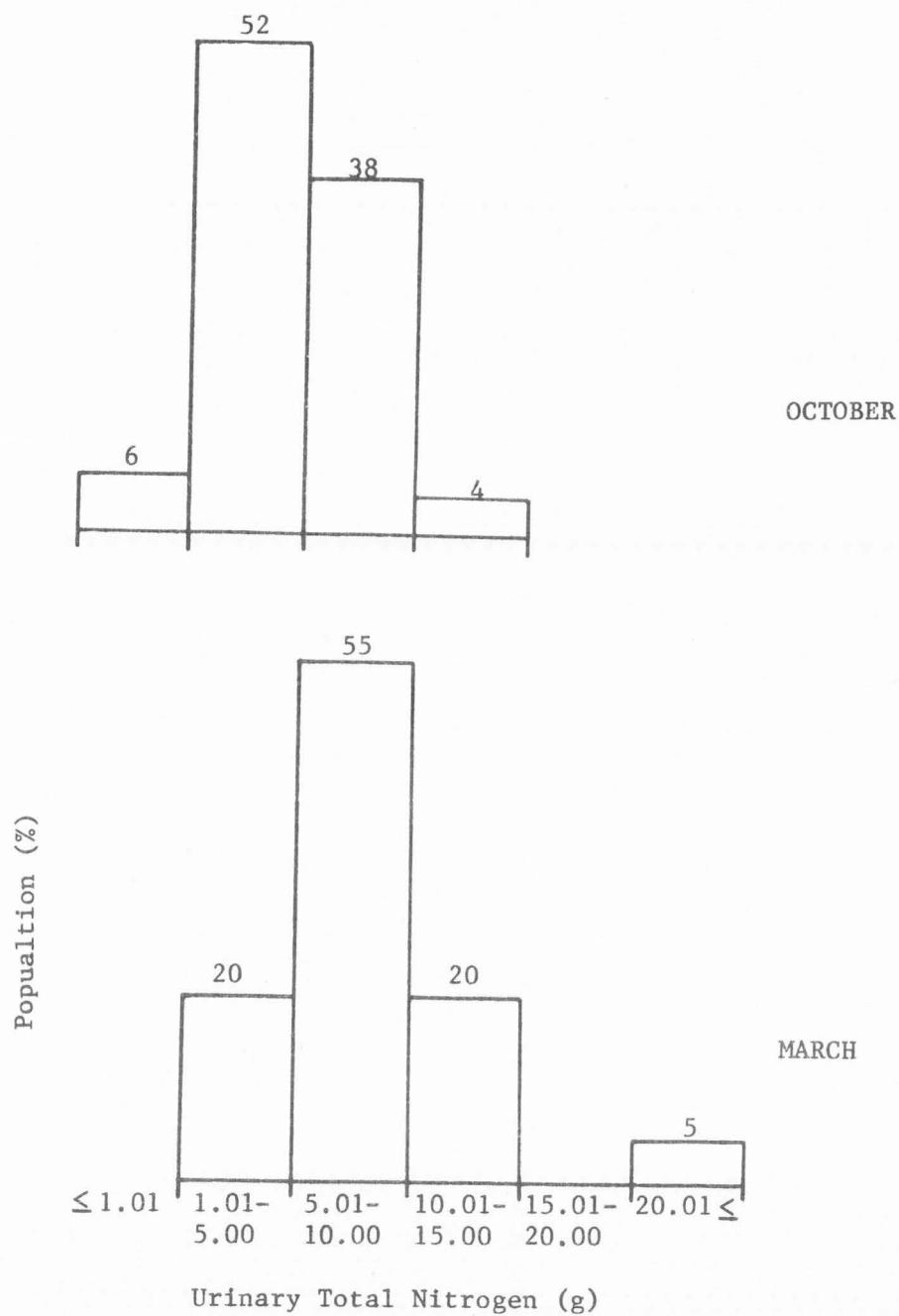


Figure 12. Frequency distribution of urinary total nitrogen excretion for women, October and March.

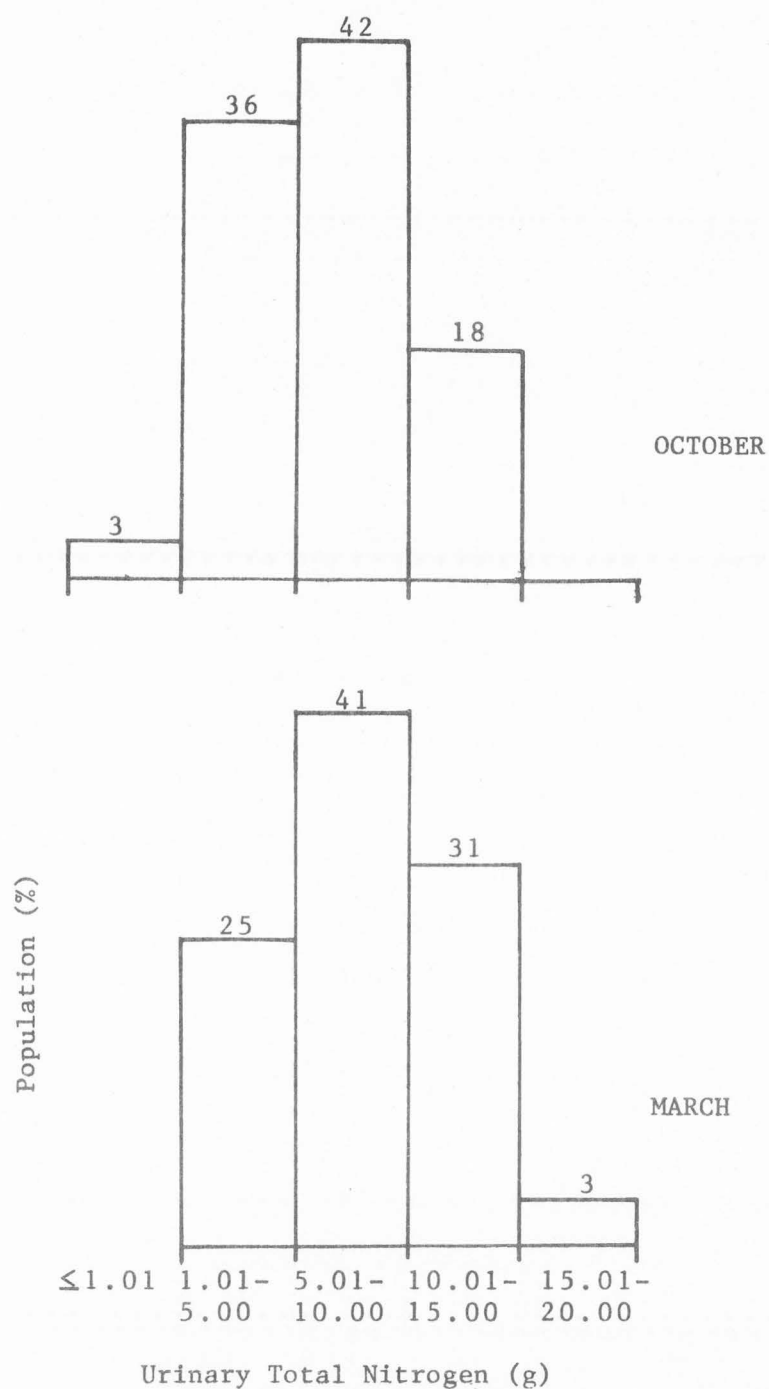


Figure 13. Frequency distribution of urinary total nitrogen excretion for men, October and March.

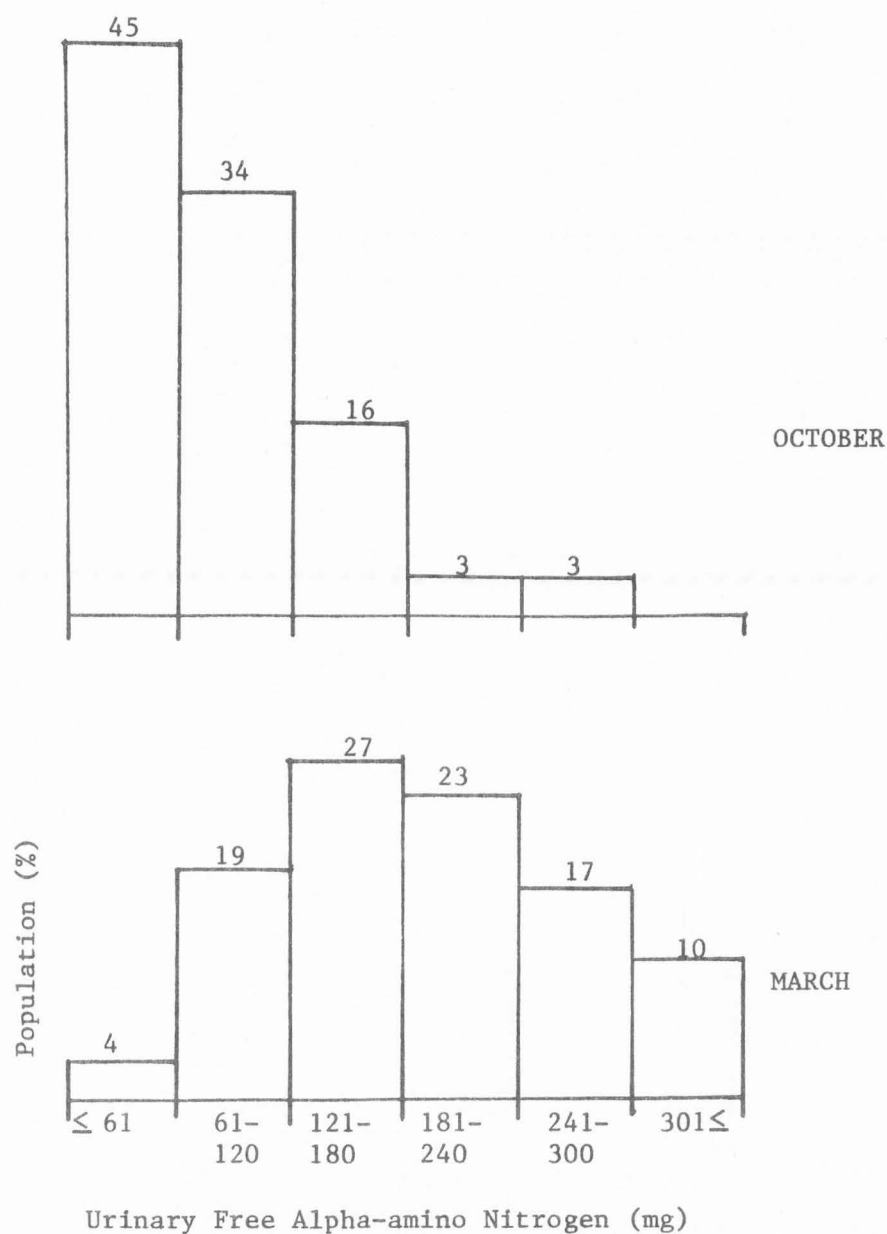


Figure 14. Frequency distribution of urinary free alpha-amino nitrogen excretion for men, October and March.

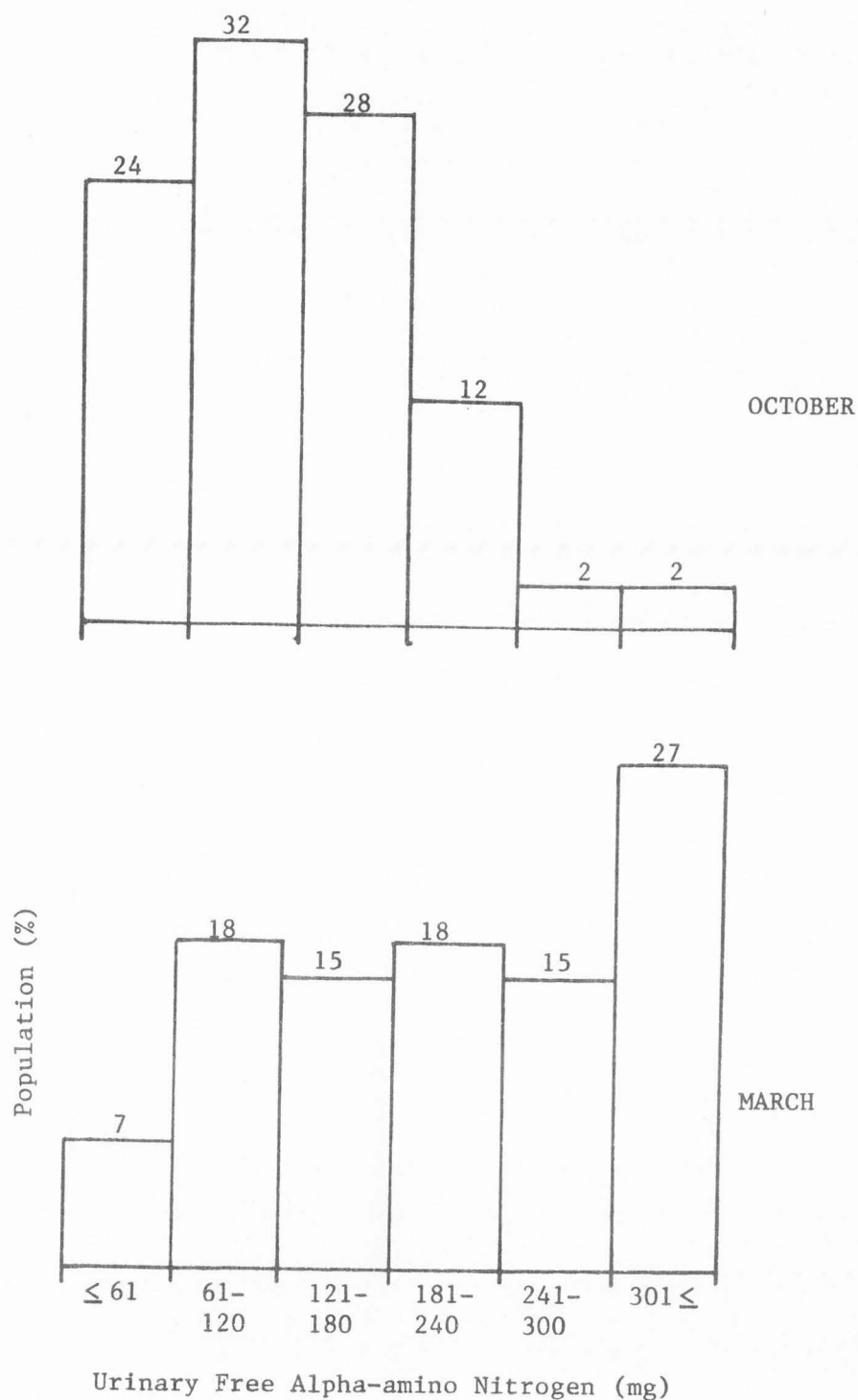


Figure 15. Frequency distribution of urinary free alpha-amino nitrogen excretion for men, October and March.

Appendix DX-rays

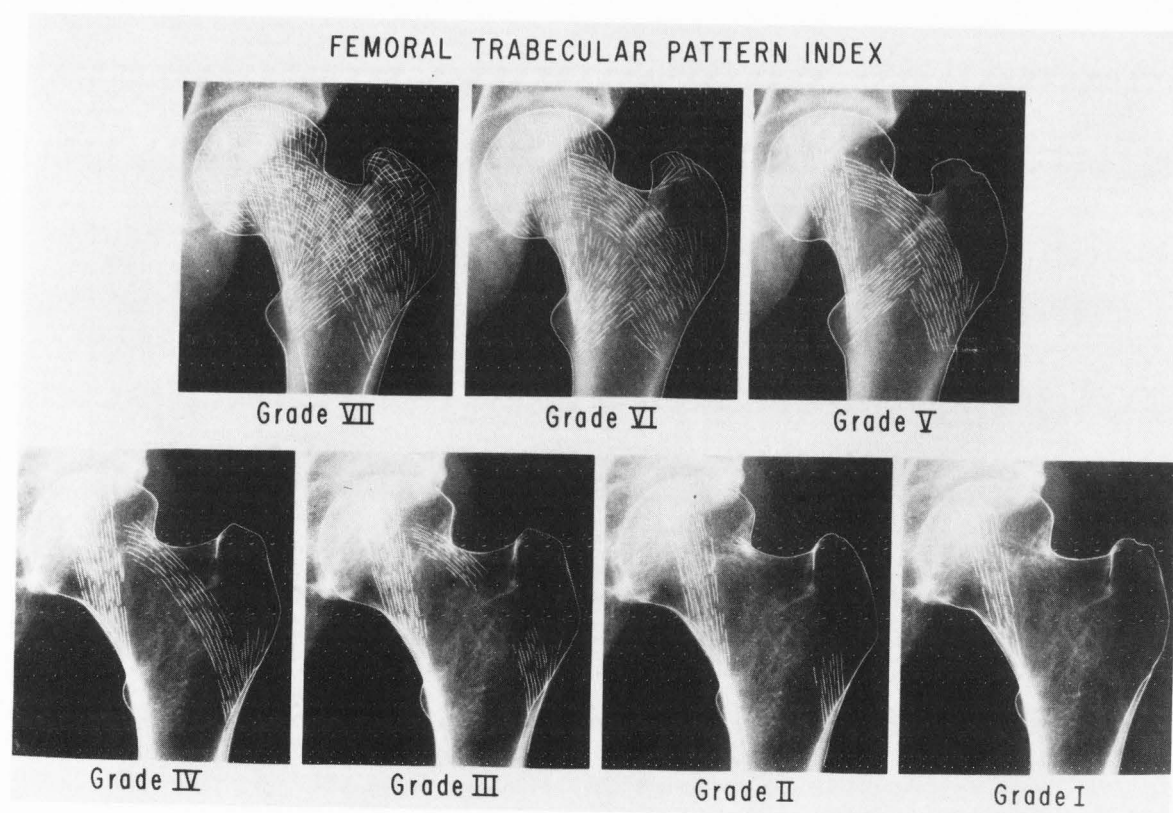


Figure 16. Trabecular pattern index.

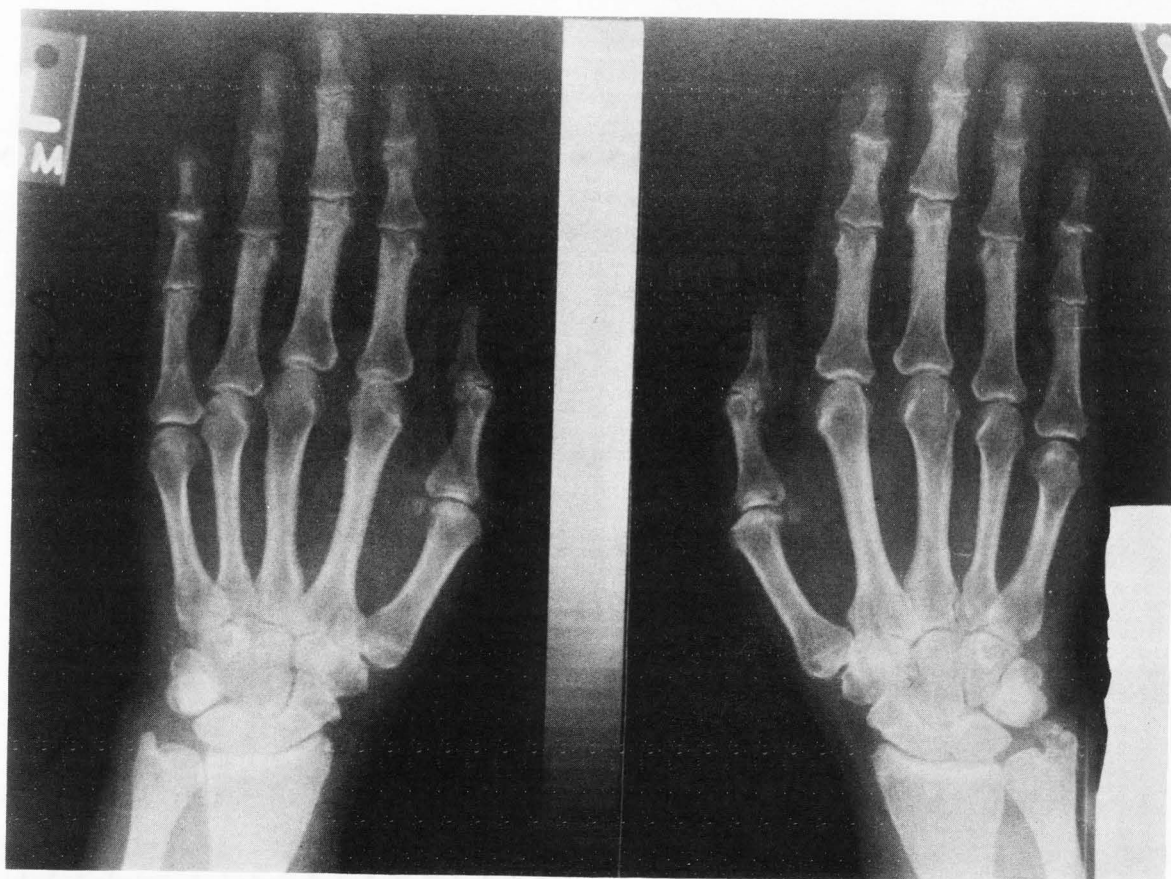


Figure 17. Normal metacarpal.



Figure 13. . Osteoporotic metacarpal.



## VITA

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